

RA1231

L 403

1940

## NATIONAL INSTITUTE OF HEALTH

H

APR 28 1941

## LIBRARY

## -- LEAD AND ITS COMPOUNDS --

(Lead) (Lead Carbonate, Basic) (Litharge)  
(White Lead, Sublimed) (Lead Arsenate)  
(Lead Sulfide) (Tetraethyl Lead)  
(Orange Mineral)

GENERAL INFORMATION

INDUSTRIAL HEALTH ASPECTS

INDUSTRIES AND OCCUPATIONS

SELECTED ABSTRACTS

SELECTED REFERENCES

OHIO DEPARTMENT OF HEALTH. *Division of Adul**Hygiene*  
R. H. MARKWITH, M.D.

Director of Health

Columbus, Ohio

1940

WITHDRAWN

from

LIBRARY

NATIONAL INSTITUTES OF HEALTH

LIBRARY

QV  
292  
9038L  
1940  
c.1

NATIONAL LIBRARY OF MEDICINE  
WASHINGTON, D. C.

RECEIVED  
NATIONAL INSTITUTE OF HEALTH  
LIBRARY  
JAN 10 1940

G-DEL 23 1959

- 2543-373 111 00100 000 -

The work of some of the earlier Greek, Roman and Arabian authors indicates that lead poisoning has been recognized for several thousand years. During the Roman era, the most common method of poisoning was the use of lead for pipes through which drinking water was conveyed. From that time to the present, medical literature has repeatedly emphasized the dangers incident to the use of this metal.

Modern studies have indicated that inhalation of lead dusts and fumes is a very important factor in lead poisoning from the ingestion of lead and its compounds. It is evident therefore that control of lead dusts and fumes is the important factor in the control of lead poisoning of industrial lead poisoning.

From the foregoing it is evident that lead poisoning is a serious and readily avoidable in the ordinary work of producing various alloys, it is caused by such large masses of pure lead and employed for such medical and important purposes. It is evident that the progress made in the elimination of such lead hazards, indicated by this work will constitute a major industrial health work.

This material compiled by the Adult Hygiene Division of the Ohio Department of Health, assisted by the personnel of Work Projects Administration in Ohio, Official Project No. 665-42-3-413.

1940

(Lead) Pb. (plumbum).

(Lead carbonate, basic)  $PbCO_3 \cdot Pb(OH)_2$ , basic lead carbonate, cerussite, lead fields, lead subcarbonate, white lead.

(Lead carbonate, PbO, lead monoxide, lead oxide (yellow), lead protoxide, plumbic oxide.

(White lead, lead(II) hydroxide).

(Lead carbonate, PbCO<sub>3</sub>).

(Lead carbonate, PbCO<sub>3</sub>, lead carbonate, white lead, lead(II) carbonate).

(Lead carbonate, PbCO<sub>3</sub>, lead carbonate, white lead, lead(II) carbonate).

(Lead carbonate, PbCO<sub>3</sub>, lead carbonate, white lead, lead(II) carbonate).

(Lead carbonate, PbCO<sub>3</sub>).

(Lead) a heavy metallic, bluish-gray, soft metal with low melting point, easily fused, malleable, ductile, and very resistant to corrosion. It is a very important metal in the manufacture of alloys, especially in the manufacture of lead-acid storage batteries. It is also used in the manufacture of lead pipes, lead shot, and lead solder.

## -- LEAD AND ITS COMPOUNDS --

The works of some of the earlier Greek, Roman and Arabian authors indicate that lead poisoning has been recognized for several thousand years. Vitruvius, the Roman architect condemned the use of lead for pipes through which drinking water was conveyed. From that time to the present medical literature has repeatedly emphasized the dangers incident to the use of this metal.

Modern studies have indicated that inhalation of lead fumes and dust is a more important factor in lead poisoning than the ingestion of lead and its compounds. It is evident therefore that control of lead fumes and dust in the atmosphere constitutes the best method of prevention of industrial lead poisoning.

From the economists standpoint it is noteworthy that no chemical, so readily capable in its ordinary uses of producing serious illness, is handled by such large numbers of persons and employed for such manifold and important purposes. In spite of the progress made in the elimination of many lead hazards, poisoning by this metal still constitutes a major industrial health hazard.

### GENERAL INFORMATION

#### CHEMICAL FORMULA AND SYNONYMS:

(Lead) Pb, plumbum.

(Lead carbonate, basic)  $2\text{PbCO}_3\text{-Pb(OH)}_2$ , basic lead carbonate, ceruse, lead flake, lead subcarbonate, white lead.

(Litharge)  $\text{PbO}$ , lead monoxide, lead oxide (yellow), lead protoxide, plumbus oxide.

(White lead, sublimed)

(Lead arsenate)  $\text{Pb(AsO}_3)_2$ .

(Lead sulfide)  $\text{PbS}$ , galena, plumbus sulfide, sulfide of lead.

(Tetraethyl lead)  $(\text{C}_2\text{H}_5)_4\text{Pb}$ , lead tetraethyl.

(Orange mineral), orange lead, sandix, orange red, saturn red.

#### PROPERTIES:

(Lead) A heavy malleable, ductile, gray, soft metal of low tensile strength, rarely found native. Sp. gr. 11.34; m.p.  $327^\circ\text{C}$ .; b.p.  $1,620^\circ\text{C}$ . Soluble in dilute nitric acid; insoluble in water. Types of lead: Chemical, soft (or common), hard, corroding.

Chemical lead. A trade term used to describe the undesilverized lead produced from the southeastern Missouri ores. According to the American Society for Testing Materials, Standard Specifications 1927, it contains from 0.04 to 0.08% copper; 0.005 to 0.015% silver, and less than 0.005% bismuth.

Soft lead. Lead of over 99 $\frac{1}{2}$ % purity.

Hard lead. Lead (originating as a by-product in lead refining) hardened or strengthened by the antimony it contains (up to 16%).

Corroding lead. A lead refined until it is sufficiently pure for the manufacture of white lead by the corroding process. The American Society for Testing Materials, Standard Specifications 1927, call for a lead containing not over 0.0015% silver, 0.0015% copper, 0.0025% copper and silver together, 0.0015% arsenic, 0.0095 antimony and tin together, 0.0015% zinc, 0.002% iron, 0.05% bismuth, and 99.933% lead by difference.

(Lead carbonate, basic) White, amorphous powder; poisonous! Sp. gr. 6.14; decomposes at 400°C. Soluble in acids; insoluble in water.

(Litharge) An oxide of lead corresponding to the formula (PbO). Soluble in alkalis and acids; insoluble in water. A fine grained canary-yellow product may contain as little as 0.05% insoluble in acetic acid and 0.005% insoluble in nitric acid. Its melting point is given variously as 776°, 879°, 888° and 907°C. Sp. gr. figures recorded range from 8.74 to 9.31 as shown by the following table:

<u>Color and Form</u>	<u>Specific Gravity</u>
Red	8.74 at 14°C.
Red, powder	9.09 at 15°C.
Red, crystals	9.125 at 14°C.
Red, crystals	9.126 at 15°C.
Yellow, crystals	9.29 at 15°C.

(White lead, sublimed) An amorphous white pigment, very uniform and fine in grain. The average composition is about as follows: Lead sulfate 75%, lead oxide 20%, zinc oxide 5%.

(Lead arsenate) White crystals; very poisonous! Sp. gr. 6.42. Soluble in nitric acid.

(Lead sulfide) Silvery, metallic crystals or black powder. Sp. gr. 7.5; m.p. 1120°C. Soluble in acids; insoluble in water and alkalis.

(Tetraethyl lead) Colorless liquid; pleasant characteristic odor. Molecular weight 323.35; sp. gr. 1.65; b.p. 152°C at 291 mm.; decomposes at 125 to 150°C. Soluble in all organic solvents. Insoluble in water and dilute acids or alkalis. Wt. per liter of vapor, 13.44 gr.

(Orange mineral) An oxide of lead corresponding to the same formula as red lead. It differs from red lead in color and in some of its properties.

#### OCCURRENCE:

(Lead) Principally in galena.

#### PREPARATION:

(Lead) By roasting and reduction principally from the ore, galena.

(Lead carbonate, basic) (a) Dutch process. By the corrosion of lead buckles in pots by means of acetic acid and carbon dioxide generated by the fermentation of waste tan-bark. (b) Carter process. By treating very finely divided lead in revolving wooden cylinders with vinegar (dilute acetic acid) and carbon dioxide. (c) Sperry process. Lead acetate is formed electrolytically and carbonated with carbon dioxide.

(Litharge) In the manufacturing process litharge may be collected in cakes of from 1 to 1.5 tons in weight when it will cool very slowly. The inner part of the cake will swell up and form flakes of red litharge, the outer part, which is necessarily chilled more rapidly, solidifies in lumps of yellow oxide. The flake may be separated from the lump by sifting and marketed as such. The solid material that remains on the screens is ground wet, settled in water and dried. This product is known as levigated litharge. The colors of the commercial grades vary from canary yellow through lemon to reddish-yellow or red, while a very pure product has the color of yellow ochre. Mechanical compression will turn the pure yellow varieties red.

(White lead, sublimed) Obtained from the sublimation of galena at a high temperature.

(Lead arsenate) By the action of a soluble lead salt on a solution of sodium arsenate, concentration, and crystallization.

(Lead sulfide) By passing hydrogen sulfide gas into an acid solution of lead nitrate.

(Tetraethyl lead) Derived by the action of ethyl halides on the various alloys of sodium and lead. Made commercially by treating a lead and sodium alloy with  $C_2H_5Cl$ .

(Orange mineral) Obtained by calcining powdered white lead.

#### IMPORTANT COMPOUNDS:

(Lead) Lead acetate, lead arsenate, lead borate, lead carbonate; lead carbonate, basic; lead chloride, lead chromate, lead dioxide,

lead nitrate; lead oxide, brown; lead oxide, red; lead silicate, lead stearate, lead sulfate, lead sulfide.

#### USES:

(Lead) Hard lead sheet is used in large quantities for roofing purposes and both hard and soft lead are used for gutters and drain pipes as well as in the chemical industries. In the manufacture, use, and handling of sulfuric acid, the use of lead for constructional purposes and equipment is practically indispensable. Lead also has a very wide-spread application in other chemical industries, as, for instance, phosphoric acid manufacture, sulfonation and chlorination processes, oil refining, gas production, and explosives industries and hydrofluoric acid handling, for lining tanks of various electroplating solutions and electrolytic refining of copper, zinc and cadmium. Some of the types of chemical equipment made from lead include pipes, flanges, valves, stirrers, agitators, tanks, coils, kettles, stills, pumps, evaporators, drying pans and drums. Lead sheet is made by rolling and pipe by extrusion. Sometimes the equipment is made from cast metal, and lead-lined or lead-covered equipment is also used, e. g., pipe, tubes, fittings, valves, coils, drums, autoclaves, tank connections, etc. Corroding lead is used for manufacturing white lead and some lead salts or compounds, however, lithargo (lead oxide) is more often used in the latter case.

Lead by itself or alloyed is an important metal for the following uses: Babbitt metal (bearing metal), type metal, storage battery grid metal, cable coverings, ammunition, solder, caulking, castings, foil, plumbing, weights, ornamental purposes and lead matrices.

(Lead carbonate, basic) Paint pigment; putty; ceramic glazes.

(Lithargo) Manufacture of storage batteries, pigments, compounding ingredient in rubber manufacture, petroleum, insecticides, varnish, linoleum, glass, pottery, and enamel.

(White lead, sublimed) It produces an excellent paint whose wearing qualities at the sea shore are superior to those of ordinary white lead and it has the additional advantage of not being so quickly acted upon by sulfur gases.

(Lead arsonate) Insecticide.

(Lead sulfide) Ceramics; preparation of metallic lead.

(Tetraethyl lead) Used for preventing knocking in internal combustion engines; certain ethylation operations.

(Orange mineral) Pigment.

## INDUSTRIAL HEALTH ASPECTS

### MODES OF ENTRANCE:

- (Lead) Inhalation and ingestion.
- (Lead carbonate, basic) Inhalation and ingestion.
- (Litharge) Inhalation and ingestion.
- (White lead, sublimed) Inhalation and ingestion.
- (Lead arsenate) Inhalation and ingestion.
- (Lead sulfide) Inhalation and ingestion.
- (Tetraethyl lead) Inhalation or absorption through the skin.
- (Orange mineral) Inhalation and ingestion.

### SYMPTOMS OF INDUSTRIAL POISONING:

Symptoms may be present as metallic taste, headache, vertigo, insomnia, easily fatigued, loss of appetite, particularly for breakfast, nausea and vomiting, obstinate constipation with occasional blood in stool and abdominal colic with appearances of a surgical belly although stated to be characteristic in that pressure usually gives relief. There may be loss of weight, blue line on the gums, nervousness, peripheral neuritis, weakness of grip, muscular twitching and tremors particularly of fingers or paralysis, especially of muscles used most or muscular cramps and pains. There may be ashen pallor, moderate anemia with increased number of cells exhibiting basophilic aggregations and stippling, also polychromatophilia, poikilocytosis, and anisocytosis. There is rarely fever, though occasionally slight jaundice, sometimes increased blood pressure, degeneration of kidneys, with hematoporphyrin in the urine. There is sometimes lead encephalopathy with irritability, excitement, delirium, convulsions and ocular disturbances as amblyopia or blindness. There may be damage to germ cells of both male and female with resultant occasional sterility, abortion, or death of fetus (still-born). Lead may be found in the blood, urine or feces.

- (Lead carbonate, basic) Symptoms of lead poisoning.
- (Litharge) Symptoms of lead poisoning.
- (White lead) Symptoms of lead poisoning.
- (Lead arsenate) Follow those of lead poisoning rather than of arsenic.
- (Lead sulfide) See lead.

(Tetraethyl lead) Symptoms are referable to the nervous system. Exposure causes insomnia, anorexia, headache, vertigo, subnormal blood pressure and temperature, restlessness, excited dreams, loss of weight, nausea and vomiting particularly in morning, marked pallor, coarse tremors, violent twitching, slow pulse rate. There may be excitement, delusions, exaggerated movements, maniacal outbursts, exhaustion, delirium, coma, convulsions and other symptoms of encephalopathy. There is anemia, stippled cells and increase lead content of blood, urine and feces. Gastric cramps, constipation or diarrhea are seldom seen.

(Orange mineral) See lead poisoning.

## INDUSTRIES AND OCCUPATIONS

INDUSTRIES: Ohio Industries using lead and its compounds as indicated in the Ohio Industrial Hygiene Survey are listed as follows:

Agricultural implements	Lead and zinc
Aircraft	Lime, cement and artificial stone
Asphalt and roofing materials	Liquor, beer, and wine
Automobile factories	Metal furniture
Blank books and paper products	Other chemicals
Blast furnaces	Other foods
Brass factories	Other manufacturing plants
Brick, tile and terra cotta	Other metal, etc.
Car and railroad shops	Other rubber factories
Chemicals	Other textiles
Copper factories	Other woodworking
Cotton cloth	Paint and varnish factories
Dairy products	Paper box factories
Dyestuffs, ink, etc.	Paper and pulp mills
Electric fixtures	Patent medicine, drugs
Electrical machinery	Petroleum refineries
Electroplating	Potteries
Embroiderie and laces	Printing
Engraving, photographic work	Rubber tires
Explosives, ammunition, fireworks	Shoes
Fertilizer factories	Soap factories
Foundries	Soft beverages
Furniture, showcases, cabinets, etc.	Storage batteries
Garages, etc.	Suits, coats and overalls
Glass factories	Textile dyeing and finishing
Hemp, jute and linen	Tin and enameled ware
Ice	Toys and unclassified novelties
Instruments	Wood, wicker, etc.
Jewelery	Woolen and worsted

OCCUPATIONS: Occupations in Ohio in which there may be contact with lead and its compounds are:

Acidulators (fertilizer factories)	Ad alley make-up men (printing)
Acid makers (fertilizer factories)	Air bag builders (rubber tires)

Annealers (other manufacturing plants)  
 Art glass workers (glass factories)  
 Assemblers (brass factories; chemicals; electric fixtures; electrical machinery; foundries; instruments; metal furniture; other manufacturing plants; other metals; storage batteries; show cases and cabinets)  
 Babbit pourers (foundries; brass factories)  
 Backers (electrical machinery)  
 Baggers (tin and enameled ware)  
 Balancers (other manufacturing plants)  
 Ball mill operators (paint and varnish factories)  
 Ball pushers (storage batteries)  
 Bead makers (rubber tires)  
 Bell inspectors (storage batteries)  
 Belt buckle makers (suits, coats & overalls)  
 Bench men (electrical machinery; foundries; instruments; optical goods; other manufacturing plants; brass factories)  
 Blacksmiths (auto factories; foundries)  
 Blockers (other manufacturing plants)  
 Bobbin makers (embroideries and laces)  
 Body builders (garages; auto factories)  
 Book binders (printing)  
 Brazers (foundries; other manufacturing plants)  
 Bridge operators (storage batteries)  
 Brush makers (brooms and brushes)  
 Buffers (electrical machinery; brass factories)  
 Bundlers (brass factories; other metal, etc.)  
 Burnishers (suits, coats, overalls)  
 Bushers (other clay, glass and stone)  
 Cabinet makers (planing and milling)  
 Car loaders (foundries)  
 Carpenters (shoes; blast furnaces; liquor, beer, & wine; planing & milling)  
 Cell liftors (storage batteries)  
 Chamferers (brass factories)  
 Chargers (storage batteries)  
 Checkers (storage batteries)  
 Chemists (storage batteries; dye-stuffs and ink; electrical machinery; lead and zinc; paint and varnish)  
 Chippers (other rubber factories)  
 Cleaners (brass factories; foundries; printing; storage batteries)  
 Coil makers (Foundries)  
 Color mixers (glass factories)  
 Commutator men (electrical machinery)  
 Compositors (paint and varnish factories; paper box factories; printing; rubber tires)  
 Compounders (dyestuffs, ink, etc.)  
 Connective operators (storage batteries)  
 Construction men (glass factory)  
 Container makers (blast furnaces; planing and milling)  
 Coro makers (brass factories)  
 Copper smiths (copper factories)  
 Copper powder makers (other manufacturing plants)  
 Cure men (rubber tires)  
 Cutters (brass factories; other manufacturing plants)  
 Die casters (brass factories; storage batteries)  
 Dippers (foundries; other manufacturing plants; potteries; storage batteries)  
 Drawers (blast furnaces)  
 Drier operators (storage batteries)  
 Dump boys (printing)  
 Electric runners (storage batteries)  
 Electricians (auto factories; coal mines; electrical machines; fertilizer factories; foundries; furniture, showcases, cabinets, etc.; glass factories; lime, cement, artificial stone; match factories; petroleum refineries; rubber tires; shoes; storage batteries; textile dyeing, and finishing; woolen and worsted)  
 Electro-platers (electro-plating; storage batteries; blast furnaces; electrical machinery; potteries; foundries)

Electrotype operators (printing)	Grinders (brass factories; metal furniture; paint & varnish factories)
Element burners (storage batteries)	Gut leaders (other manufacturing plants)
Element setters (storage batteries)	Hardeners (foundries)
Enamelers (foundries; electrical machinery; jewelry; tin and enameled ware)	Heat treaters (auto factories; foundries; printing)
Engineers (car and railroad shops; foundries; storage batteries; electrical machinery)	Inspectors (brass factories; electrical batteries)
Engravers (printing; blank book and paper products; other manufacturing plants)	Intertype operators (printing)
Explosive makers (explosives, ammunition, fireworks)	Janitors (printing; storage batteries; tin and enameled ware)
Filers (brass factories; metal furniture)	Jewelers (jewelry)
Fillers (foundries; other metals, etc.; paint & varnish factories; wood, wicker, etc.)	Kettle men (chemicals)
Finishers (brass factories; auto factories; metal furniture; engraving and photographic work; printing; wood, wicker, etc.; electrical machinery; other manufacturing plants)	Laborers (blast furnaces; brass factories; dye stuffs, ink, etc.; foundries; suits, coats, overalls; tin & enamel ware; electrical machinery; lead & zinc; metal furniture; paint & varnish factories; storage batteries)
Firemen (foundries; other rubber factories)	Lathe operators (brass factories; foundries)
Floor men (engraving, photographic work; storage batteries)	Lay-out men (foundries; metal furniture)
Floor washers (storage batteries)	Lead burners (auto factories; blast furnaces; fertilizer factories; soap factories; storage batteries)
Foremen (fertilizer factories; garages; lead & zinc; metal furniture; suits, coats, overalls; paint & varnish factories; potteries; printing; storage batteries; blank book & paper products; brass factories; chemicals; dyestuffs, ink, etc.)	Lead coaters (brass factories)
Frame makers (foundries)	Leaders (other manufacturing plants)
Frit enamel makers (tin & enameled ware)	Lead heaters (foundries)
Furnace tenders (brass factories; lead & zinc; storage batteries)	Lead insulators (rubber tires)
General helpers (liquor, beer & wine)	Lead strippers (rubber tires)
General sign painters (electrical machinery)	Lead type tubers (printing)
Generator men (storage batteries)	Lift-off men (storage batteries)
Glazo makers (brick, tile & terracotta; potteries)	Linotype operators (electrical machinery; other woodworking; printing)
Glazers (glass factories; other clay, glass and stone; planing & milling)	Lock-up men (printing)
	Lubricator testers (brass factories)
	Lug brushers (storage batteries)
	Machine operators (printing; storage batteries; electrical machinery)
	Machinists (foundries; other clay, glass & stone; printing; rubber tires; shoes; storage batteries; suits, coats, overalls; blank book & paper products; blast furnaces; brass factories; electrical machinery)

Maintenance men (chemicals; electrical machinery; foundries; other foods; soap factories)

Make-up men (printing)

Mat-makers (printing)

Mechanics (dairy products; fertilizer factories; foundries; garages; ice; printing; shirts, collars, cuffs; soft beverages; storage batteries; suits, coats, overalls; aircraft)

Melters (brass factories; paint & varnish factories; printing; other rubber factories)

Metal workers (foundries; furniture, showcases, cabinets, etc.; other manufacturing plants; other metal, etc.)

Metallurgists (foundries)

Meter men (storage batteries)

Mill men (rubber tires; dyestuffs, ink; paints & varnish; brass factories)

Millwrights (electrical machinery; furniture; showcases, cabinets, etc.; paint & varnish factories)

Mixers (chemicals; other clay, glass and stone; paints & varnish; rubber tires; glass factories; dyestuffs & ink; storage batteries; asphalt & roofing materials; other rubber factories; tin and enameled ware)

Model men (electrical machinery)

Mold cleaners (storage batteries)

Mold makers (storage batteries)

Mold setters (storage batteries)

Molders (brass factories; car & railroad shops; electrical machinery; foundries)

Monotype operators (engraving, photographic work; printing)

Mounters (other manufacturing plants)

Multigraph operators (other metal, etc.)

Office workers (printing)

Oilers (blast furnaces; storage batteries)

Oxide supply men (storage batteries)

Packers (other manufacturing plants; paint & varnish factories; potteries; soap factories; storage batteries; blast furnaces;

electrical machinery; lead and zinc)

Painters (car & railroad shops; liquor, beer and wine; foundries; furniture, showcases, cabinets, etc.; garages; ice; other manufacturing plants; other woodworking; printing; agricultural implements; auto factories; blast furnaces; brass factories; wood, wicker, etc.; rubber tires; storage batteries; toys & unclassified novelties)

Paint mixers (foundries; other chemicals; paint & varnish factories)

Paper bailers (storage batteries)

Paper rulers (printing)

Paste car washers (storage batteries)

Paste mixers (storage batteries)

Pasters (storage batteries)

Pattern makers (blast furnaces; brass factories; foundries; electrical machinery; other wood working; blank book and paper products)

Picklers (brass factories)

Pig lead feeders (storage batteries)

Pipe fitters (electrical machinery; foundries; glass factories; paint & varnish factories; petroleum refineries; rubber tires; soap factories; storage batteries; blast furnaces; chemicals; dyestuffs, ink, etc.; brass factories; textile dyeing and finishing; printing; woollens & worsted; match factories; paper box factories)

Planers (printing)

Plasterers (storage batteries)

Plate castors (printing; suits, coats and overalls)

Polishers (brass factories; marble & stone; metal furniture)

Press feeders (printing; suits, coats and overalls)

Press men (electrical machinery; lead and zinc; paint & varnish factories; printing; rubber tires; auto manufacturing plants; brass factories; storage batteries)

Printers (other woodworking; paint & varnish factories; printing; suits, coats & overalls; blank book & paper products; hemp, jute & linen; jewelry; paper & pulp mills)

Process men (fertilizer factories)  
Pump men (blast furnaces; storage batteries)  
Putty makers (paint & varnish factories)  
Rackers (storage batteries)  
Radio engineers (electrical machinery; storage batteries)  
Receivers (brass factories; storage batteries; other rubber factories; rubber tires; lead & zinc)  
Reelers (blast furnaces)  
Refiners (storage batteries)  
Repairmen (brass factories; storage batteries; foundries; toys & unclassified novelties; garages; agricultural implements; auto factories; chemicals; electrical machinery)  
Riggers (blast furnaces)  
Rubber cutters (other rubber factories)  
Safety equipment men (storage batteries)  
Sagger washers (potteries)  
Salvagers (explosives, ammunition, fireworks)  
Sample boys (storage batteries)  
Sand plasterers (brass factories; foundries; storage batteries)  
Sand screeners (brass factories)  
Saw operators (storage batteries; brass factories)  
Screw cutters (brass factories)  
Separators (brass factories; storage batteries)  
Set-up men (brass factories)  
Shake-out men (brass factories)  
Shavers (brass factories)  
Shear operators (brass factories)  
Shoot metal workers (blast furnaces; brass factories; copper factories; wood, wicker, etc.; electrical machinery; foundries; furniture, showcases, cabinets, etc.; instruments; metal furniture; tin & enameled ware)  
Shipping clerks (brass factories; lead & zinc; potteries; storage batteries)  
Shovel operators (tin & enameled ware)

Sign writers (other manufacturing plants)  
Skat tower operators (explosives, ammunition, fireworks)  
Smelter operators (printing; tin & enameled ware)  
Snail tiers (other manufacturing plants)  
Solderers (brass factories; electric fixtures; electrical machinery; foundries; other manufacturing plants; other metal, etc.; storage batteries)  
Sorters (other metals; storage batteries)  
Spinners (electric fixtures; other manufacturing plants)  
Sprayers (electric fixtures; other manufacturing plants; potteries; tents & awnings; brick, tile, and terra cotta; car & railroad shops; foundries)  
Stamp men (jewelry)  
Stamp mounters (other rubber factories)  
Station attendants (storage batteries)  
Stencil men (storage batteries)  
Stereotype operators (blank book & paper products; printing)  
Stonemen (paint & varnish factories)  
Storage men (chemicals)  
Stove control assemblers (electrical machinery)  
Strappers (storage batteries)  
Strippers (electrical machinery)  
Superintendents (brass factories; fertilizer factories; storage batteries)  
Supervisors (printing; rubber tires; storage batteries)  
Supply men (storage batteries)  
Surfacers (metal furniture)  
Sweepers (brass factories; storage batteries)  
Switch operators (storage batteries)  
Take-off men (storage batteries)  
Telephone men (blast furnaces)  
Temperers (foundries)  
Template makers (rubber tires)  
Terminal riveters (electrical machinery)  
Testers (blast furnaces; electrical

machinery; storage batteries)  
Thinners (paint & varnish factories)  
Tinnern (electrical machinery; found-  
ries; glass factories; match fac-  
tories; ship & boat building; stor-  
age batteries; tin & enameled ware;  
other textiles; other woodworking;  
planing & milling; rubber tires)  
Tinsmiths (electrical machinery; soap  
factories; suits, coats & overalls;  
woolen & worsted)  
Tool makers (storage batteries)  
Touch-up men (storage batteries)  
Trade mark fillers (other manufac-  
turing plants)  
Trimmers (electric fixtures; metal  
furniture; suits, coats & overalls)  
Trouble shooters (electrical machin-  
ery; storage batteries)  
Truckers (brass factories; storage  
batteries)  
Tumblers (brass factories)  
Type casters (printing)  
Type setters (instruments; other manu-  
facturing plants; printing; electri-  
cal machinery; paint & varnish fac-  
tories; paper box factories; other  
rubber factories; rubber tires;  
blank book & paper products; cotton  
cloth; engraving, photographic  
work)

Type washers (printing)  
Unloaders (car & railroad shops;  
storage batteries)  
Utility men (fertilizer factories;  
rubber tires)  
Varnish makers (paint & varnish  
factories)  
Vise men (foundries)  
Warehouse men (dyestuffs, ink;  
foundries; lead & zinc)  
Washers (storage batteries)  
Weighers (tin & enameled ware;  
rubber tires)  
Welders (auto factories; metal  
furniture; rubber tires;  
foundries; furniture, show-  
cases, cabinets, etc.; garages)  
Wheel dressers (lead & zinc)  
Winders (lead & zinc; blast  
furnaces; electrical machinery)  
Wipers (blast furnaces; found-  
ries)  
Wood workers (wood, wicker, etc.)

## LEAD HAZARDS IN OHIO INDUSTRIES

The listing of occupations and industries in the preceding pages in which contact with lead and its compounds is indicated was based on an inventory of materials used in various industrial processes. It does not imply that a harmful exposure to lead exists in every case. The degree of harmfulness of an exposure, however, may be ascertained by engineering studies of the industrial environment. The studies of numerous investigators, who have correlated chemical and engineering findings with physical and statistical studies upon the exposed workers, have made possible certain standards of exposure to harmful materials. A standard widely accepted for exposure to lead is 1.5 milligrams of lead per 10 cubic meters of air. If the concentration of air borne lead exceeds this value for a prolonged period of time symptoms of lead poisoning may develop. As would be expected the danger of lead poisoning increases with the increase of the amount of lead in the air.

The Ohio Department of Health has collected and analyzed air samples for their lead content in various types of industrial establishments. A summary of these determinations made in certain industrial groups is presented on the next page to show that real lead hazards prevail in Ohio Industries. It should be emphasized that all determinations made in the types of industries listed are included in the table regardless of whether the concentrations found were high or low. Studies made in automobile factories are divided into two groups, those in which uncontrolled exposures prevailed and those in which adequate ventilation facilities prevented dangerous concentrations of lead in the air. This clearly demonstrates that serious industrial health hazards may be eliminated by the institution of proper control measures.

Methods which may be used in the effective control of lead hazards in industry depend in a large part upon the conditions under which the lead is used. The primary objective of any successful control program is the elimination of exposure to dangerous concentrations of lead in the workers' environment. In many cases it may be necessary to examine the worker as well as his environment in order to successfully evaluate the lead exposure. Thorough physical examinations accompanied by blood and urine tests are necessary in exhaustive studies. In routine check-ups blood examination alone may be indicative of the individual's response to the lead in his environment. In order to encourage and assist industry and physicians in instituting and carrying out a program of routine blood examinations, the Ohio Department of Health has prepared a bulletin "The Basophilic Aggregation Test for Lead Absorption and Lead Poisoning". This bulletin may be obtained upon request from the Ohio Department of Health, R.H. Markwith, M.D., Director.

DISTRIBUTION OF ATMOSPHERIC LEAD DETERMINATIONS, MADE BY THE OHIO  
DEPARTMENT OF HEALTH, ARRANGED ACCORDING TO NUMBER OF SAMPLES  
IN SPECIFIED INDUSTRIES FOUND IN EACH CONCENTRATION GROUP.

INDUSTRIAL CLASSIFICATION	Concentration Groups in Milligrams of Lead per 10 Cubic Meters of Air.						
	Below 1.5	1.5-4.9	5.0-9.9	10-24.9	25-49.9	50-100	Above 100
Automobile Factories (Uncontrolled Exposures)	13	4	13	22	10	11	4
Automobile Factories (Controlled Exposures)	14	-	-	-	-	-	-
Brass and Bronze Factories	25	39	26	20	6	1	-
Electrical Machinery	6	-	-	-	-	-	-
Glass	12	47	23	10	1	1	1
Metal Furniture	4	-	-	-	-	-	-
Potteries	2	1	2	2	-	1	-
Storage Batteries	7	12	5	8	-	-	-
Tin and Enamelware	4	15	8	5	4	3	4
Welding, Forging and Heat Treating	21	4	6	5	3	1	-
TOTAL (All Industries)	. 108	. 122	. 83	. 72	. 24	. 18	. 9

## SELECTED ABSTRACTS

### DETERMINATION OF LEAD IN THE AIR.

Air Hygiene Foundation of America, Inc.

Preventive Eng. Ser. Bull. No. 2, Pt. 6, 9 pp. (1938).

Chemical Abstracts, vol. 33, p. 75.

For Pb dusts and mists (as in spray coating) an impinger can be used to collect the sample from 1 cu.ft. of air per min. in about 75 cc. of distd. water or 10%  $\text{HNO}_3$ ; for Pb fumes, the elec. precipitator is preferable. Usually 30-45 cu.ft. of air should be taken as samples and all glassware, reagents and distd. water employed should be free from and regularly tested for Pb. The following analytical procedures are satisfactory for routine detns. of Pb in collected air samples: (1) Volumetric chromate method.--Pb is freed of interfering materials and pptd. as chromate. Indirect detns. of Pb is made by soln. and iodometric estn. of the chromate ion. It is best to have 0.1 mg. or more of Pb in the sample which amt. will be present in 30 cu.ft. of air when the concn. is about 1.5 mg. per 10 cu.m. If the concn. is considerably above or below that amt. then smaller or larger samples should be taken. (2) Colorimetric dithizone method.--Pb is extd. from a soln. of controlled alky. with a  $\text{CHCl}_3$  soln. of dithizone (diphenylthiocarbazone) which forms colored metallo-org. compds. with several heavy metal ions. By proper control of alky. and cyanide concn., interfering ions are eliminated and the cherry-red Pb compd. is segregated in soln., the intensity of color depending on the amt. of Pb present. For detn. the color is compared with that of dithizone solns. contg. known amts. of Pb. The quantity of Pb in the portion of the sample taken for analysis should be less than 0.1 mg. and preferably not over half that much. This allows sampling of quantities of air smaller than 30 cu.ft., particularly if the Pb concn. is suspected of being above 1.5 mg. per cu.m. (3) Microscopic detection.--The presence or absence of Pb in dust samples, in raw materials such as paint pigments, glazes, enamels, etc., or in excreta can be detected by a microscopic method in which the  $\text{PbI}_2$  is pptd. from a drop of soln. and identified microscopically by its form and color. Sb, Bi and Ag interfere with this detection and if present must be eliminated by special methods. App., reagents, solns., procedures, calens., equations and typical examples are described in detail and photomicrographs are given for  $\text{Pb}(\text{NO}_3)_2$  and  $\text{PbI}_2$  crystals.

### METHODS FOR THE ANALYSIS OF DUST AND FUMES FOR LEAD AND ZINC.

S. Moskowitz and Wm. J. Burke.

J. Ind. Hyg. Toxicol. 20, 457-64 (1938).

Chemical Abstracts, vol. 33, p. 76.

Dust samples are evapd. to dryness in the presence of  $\text{HNO}_3$ , the residue extd. with  $\text{HCl}$  and the Pb + Zn + Pb detd. by the dithizone method. Accuracy and precision of the method is 0.001 mg. when less than 0.050 mg. of the metals is present.

### DETERMINATION OF LEAD BY DITHIZONE, MODIFICATIONS AND IMPROVEMENTS OF THE HUBBARD-CLIFFORD-WICHMANN METHOD AS APPLIED TO BIOLOGICAL MATERIAL.

K. Bambach. Indust. Eng. Chem., Anal. Ed., vol. 11, pp. 400-403 (July 15, 1939).

Abstracted in J. of Ind. Hygiene, vol. 21, no. 9, p. 218 (abstract section) Nov. 1939.

The following modifications and improvements have been made in the dithizone method for the determination of lead in biological material described by Hubbard:

The use of hydroxylamine in the initial extraction prevents the oxidation of the dithizone and permits the elimination of the second extraction step of the Hubbard method.

Washing the first chloroform extract removes extraneous extraneous salts and improves the test for bismuth.

Filtrations through cotton and through paper have been eliminated from the procedure without loss of accuracy, and with a resultant saving of time and decrease of opportunities for contamination.

The addition of the standard dithizone solutions and the development of the mixed color in all the samples of a series before the photometric readings are made save time and do not affect the accuracy of the analyses.  
--Author's summary.

#### LEAD IN HUMAN TISSUES.

K.N. Bagehi, H.D. Ganguly, and J.N. Sirdar. Indian J. Med. Res., vol. 26, pp. 935-945 (Apr. 1939).

Abstracted in J. of Ind. Hygiene vol. 21, no. 8, pp. 191-192 (abstract section) Oct. 1939.

Examination of lead by the dithizone method was carried out on a series of normal persons as well as on persons with known exposures to lead. The figures for normal lead are roughly similar to Kehoe's but are only a fraction of those reported by Tompsett and Anderson. Amounts in different organs of exposed persons was high in the liver, kidneys, stomach and intestines and exceptionally high in the hair. Evidently hair of different colors absorbs differing amounts of Pb; this is to be reported on later. (No figures are given as to concentrations the patients were exposed to or the degree of poisoning.) Lead in tissues of fetuses is smaller than that reported by other writers. No trace could be found in ovaries although Pb was found in testes and placentas.--Helen Lawson.

#### THE LEAD CONTENT OF HUMAN BLOOD.

C.E. Willoughby and E.S. Wilkins, Jr. J. Biol. Chem., vol. 124, pp. 639-657 (Aug. 1938).

Abstracted in J. of Ind. Hygiene, vol. 20, no. 9, pp. 202-203 (abstract section) Nov. 1938.

Blood specimens from 189 individuals with histories showing no abnormal lead exposure were analyzed for lead, using the dithizone method. The values ranged from 0.00 to 0.09 mgm. Pb per 100 gm. blood, with a most probable value of  $0.025 \pm 0.002$  mgm. This is significantly lower than the most probable value of previous investigators.

Lead was found to be absent from the serum in 90% of the 58 samples

in which clot and serum were analyzed separately, contrary to the findings of Blumberg and Scott, and Teisinger.

Blood lead values as high as 0.1 mg.% may occur without indisputable clinical evidence of plumbism.--G.H. Hitchings.

#### EXAMINATION OF LEAD IN CEREBROSPINAL FLUID OF NORMAL AND LEAD POISONED PERSONS.

F. Schmitt and W. Basse. Klin. Wehnschr., vol. 16, pp. 65-66 (1937).

Abstracted in J. of Ind. Hygiene, vol. 19, no. 4, p. 84 (abstract section) April 1937.

In the cerebrospinal fluid of 13 normal persons, the authors found 15-38 micrograms % of lead; in 5 cases of lead poisoning the amounts were 80-493 micrograms %. The amount of lead in the spinal fluid does not parallel blood lead.--L. Teloky.

#### THE BASOPHILIC AGGREGATION TEST FOR LEAD POISONING AND LEAD ABSORPTION; TEN YEARS AFTER ITS FIRST USE.

C.P. McCord, F.R. Holden, and J. Johnston. Indust. Med., Apr., 1935, vol. 6, pp. 180-185.

Abstracted in J. of Ind. Hygiene, vol. 17, no. 5, p. 97, (abstract section) Sept. 1935.

A need long has existed for a simple laboratory test serving as an index of lead absorption and the imminence of lead poisoning. An approach to such a test was described by us in 1924. Through technical improvements made by others and by us, this procedure is now suited to application by any physician or laboratory doing blood work. The basis of the test is the enumeration of the totality of erythrocytes containing basophilic substance rather than qualitative or quantitative examinations for stippled cells or polychromatophilic cells.

The native state of basophilic substance in unaltered blood is unknown but by taking the red cells this substance is artificially aggregated into readily visible masses. In normal human adults these aggregations rarely exceed 1% of the total number of erythrocytes but in lead exposed individuals the percentage frequently lies above this normal maximum. Findings of percentages above 1 to 1.5% and especially above 2% in persons exposed to lead at once suggest lead absorption and the possibility of approaching lead poisoning.

The chief value of this test is as a mark of lead absorption and early lead poisoning. As lead poisoning progresses to extended chronicity, the worth of the procedure diminishes.--Authors' summary.

#### COMPARISON OF PUNCTATE BASOPHILIA AND RATIO OF LARGE TO SMALL LYMPHOCYTES IN THE DIAGNOSIS AND PREVENTION OF LEAD POISONING.

D.O. Shiels. M.J. Australia, vol. 1, pp. 535-545 (Apr. 10, 1937).

Abstracted in J. of Ind. Hygiene, vol. 19, no. 10, p. 224 (abstract section) Dec. 1937.

This is a very careful study of laboratory tests in regard to the diagnosis of lead poisoning. The stippled cell count is shown to give

false impressions of the clinical condition. It may even rise as the patient improves. The author lays greater stress on the ratio of large to small lymphocytes in the blood which, he says, is more closely correlated with the clinical condition than is stippling. Urinary lead examinations were also made. By correlating this vast amount of data, the conclusion is reached that it is inadvisable to adjudge the severity of cases by laboratory standards alone. Evidence of lead poisoning with disability is to be expected when the laboratory data show (a) total stippled count 2,500; (b) coarsely stippled cell count, 1,000; (c) ratio of large to small lymphoid cells, less than 1.5; (d) concentration of lead in urine, 0.15 mgm./L. and over; (e) coarsely stippled cell count divided by the ratio of large to small lymphocytes, 800.--J.C. Aub.

#### THE CLINICAL SIGNIFICANCE OF PUNCTATE BASOPHILIA IN THE ERYTHROCYTE.

E.H. Falconer. Ann. Int. Med., vol. 12, pp. 1429-1441 (Mar. 1939).

Abstracted in J. of Ind. Hygiene, vol. 21, no. 6, p. 143 (abstract section) June 1939.

The mean number of stippled erythrocytes in 205 normal adults was  $92 \pm 4.4$  (P.E. of mean) per million red cells; in 234 patients with different clinical conditions, it was 363 per million. Five patients, to whom lead was administered, had 19,950,  $\pm 837$  per million after symptoms appeared, but 2250,  $\pm 1115$  up to the time symptoms appeared. In 8 workers exposed to lead but free of symptoms, the stippled cell count was 2100. Administration of alkaline hematin to 2 patients resulted in stippled cell counts of 5250 and 2092.

These data suggest that punctate basophilia is not a specific indication of either lead absorption or lead intoxication.--George Saslow.

#### CONCERNING THE NATURALLY OCCURRING PORPHYRINS. IV. THE URINARY PORPHYRIN IN LEAD POISONING AS CONTRASTED WITH THAT EXCRETED NORMALLY AND IN OTHER DISEASES.

D.J. Watson. J. Clin. Invest., vol. 15, pp. 327-334 (May, 1936).

Abstracted in J. of Ind. Hygiene, vol. 18, no. 7, p. 99 (abstract section) Sept. 1936.

Coproporphyrin I has been isolated from the urine of a normal individual. It is probable that relatively very small amounts of coproporphyrin III were likewise present.

Coproporphrin I was obtained in much larger amount from the urine of an individual with fever due to pulmonary suppuration, and from a patient with hemolytic jaundice during a postoperative "hemoclastic" crisis. In both of the latter instances considerable increases in the urine urobilinogen were observed simultaneously with the heightened excretion of coproporphyrin. A porphyrin was isolated from the urine in three cases of lead poisoning. The crystal form and melting point of the ester indicated clearly that this was, in each instance, coproporphyrin III.--Author's summary.

#### EARLY IDENTIFICATION OF SATURNISM BY THE DETERMINATION OF PORPHYRIN BY FIKENTSCHER AND FRANK'S METHOD.

H. Otto. Arch. Gewerbepath. Gewerbehyg. 8, 655-60 (1938).

Chimie & industrie 40, 1097.  
Chemical Abstracts, vol. 33, p. 2608.

Observations on 130 Pb workers showed that detn. of porphyrin provides a good method of detecting Pb poisoning at an early stage. It is preferable to hematological examn. Workers after employment for 2 years in a dangerous occupation, showed a high porphyrin content. The test will reveal what workers are in danger of becoming Pb poisoned. The method used consists in detecting and evaluating, in ultraviolet light, the red fluorescence produced by porphyrin extd. from 30 cc. of urine by means of EtOAc.

#### EFFICIENCY AND RANGE OF THE TIME-STIMULUS METHOD (CHRONAXIE) AND THE BASOPHILIC STIPPLED ERYTHROCYTE COUNT IN INDUSTRIAL HYGIENE.

F.H. Lewy. Arch. f. Gowerbepathol. u. Gowerbehyg., 1935, vol. 6, pp. 63-69.

Abstracted in J. of Ind. Hygiene, vol. 17, no. 5, p. 96 (abstract section) Sept. 1935.

The author notes the difference between the clinical-medical observation of a single patient and mass observations made on industrial cases. Similar observations on 825 lead workers showed that chronaxie and blood observations complement each other admirably. In a heavy exposure, the stippled erythrocyte count rises above normal in a single week, and falls again quickly after the exposure ceases. On the other hand, changes in nerve-muscle irritability occur only after some months' exposure and never, even after months and years of freedom from exposure, return to normal. Blood counts show to better advantage the immediate danger, and chronaxie the danger which the industry as a whole provides. Besides there are some lead workers who show only one of the two reactions. (See This Journal, May, 1935, pp. 73 and 79)--L. Teleky.

#### THE DANGER OF EMPHASIZING LABORATORY FINDINGS IN LEAD POISONING DIAGNOSIS.

M.R. Mayers. N.Y. State Dept. Lab., Indust. Bull., vol. 16, pp. 289-290 (July, 1937).

Abstracted in J. of Ind. Hygiene, vol. 19, no. 10, p. 225 (abstract section) Dec. 1937.

This is a plea for a greater attention to clinical diagnosis instead of depending too much on laboratory findings which may be expensive and often inconclusive. There are no specific criteria for making a positive diagnosis in lead poisoning or any other disease.--Helen Lawson.

#### THE RETENTION AND ELIMINATION OF LEAD.

J.C. Aub and A. Minot. Proc. Am. Soc. Clin. Investigation, Jour. Am. Med. Assn., June 2, 1923, vol. 80, p. 1643.

Abstracted in J. of Ind. Hygiene, vol. 5, no. 6, pp. 120-121 (abstract section) Oct. 1923.

"After lead has been absorbed through the gastro-intestinal or respiratory tracts, it is stored nearly completely in the calcareous portion of the bones. Here it remains normally for long periods, with the gradual release of only small amounts. In one group of animals the esophagus was tied and lead carbonate was introduced directly into the lungs. The animals were given physiologic sodium chlorid solution intraperitoneally. When the

animals were killed, within eighty hours after the injection, nearly all of the absorbed lead was found in the bones. In animals allowed to live between eighty and 160 hours, only about 81 per cent of the lead was found in the bones. When sodium bicarbonate instead of salt solution was injected intraperitoneally, the amount of lead found in the bones did not diminish, but remained at 94 per cent of the total absorbed lead. This strongly suggested that the reduction of the alkali reserve allowed the liberation of lead from the bones. We next found that in loaded cats starvation will cause the lead to become distributed throughout the organism and be excreted with increased rapidity. Ingestion of large amounts of phosphoric acid has a similar but more marked effect. Using these observations in treating lead poisoning in man, we have found that ingestion of acid causes a markedly increased excretion of lead by the urine and feces. Phosphoric acid is the most effective method so far found. It is more efficient than potassium iodid, and causes a fourfold increase in the rate of lead excretion over that occurring without treatment.

"These experiments explain how distortion of the acid-base equilibrium may liberate lead from the bones long after its absorption; they explain why lead poisoning is a chronic disease with possible late acute manifestations, and they offer a new method for increasing or decreasing the excretion of lead."--K.R.D.

#### ELIMINATION OF LEAD IN URINE OF HUMANS AFTER SINGLE ORAL DOSES.

T. Ohmura. Ztschr. f. d. ges. exp. Med., vol. 98, pp. 769-771 (1936).

Abstracted in J. of Ind. Hygiene, vol. 18, no. 9, pp. 163-164 (abstract section) Nov. 1936.

Lead elimination in research subjects normally was 4-10 micrograms a day, but after a dose of 10 mg. lead chloride, it rose to about 90 micrograms on the 1st and 2nd day, then immediately decreased from the 3rd day until it was 20-30 micrograms at the end of 10 days; on the 25th day it was about 15 micrograms. Up to the 23rd day only about 4% was eliminated (the author forgets to add "in the urine"--the feces he did not consider).

Independently of this experiment, the suggestion has come from the Berlin Pharmacological Institute that after cessation of metal dosage, the faster the poisoning occurs the more quickly the lead elimination will diminish. High lead elimination, which, after the lapse of a few days, suddenly diminishes, permits the conclusion that the subject had taken a large quantity of lead immediately before the examination.--L. Teleky.

#### LEAD MOBILIZATION AND MINERAL METABOLISM IN LEAD POISONED PERSONS.

F. Schmitt and H. Taeger. Ztschr. f. exp. Med., vol. 101, pp. 21-41 (1937); see also authors' abstract in Arbeitsschutz, pp. 176-177 (1937).

Abstracted in J. of Ind. Hygiene, vol. 19, no. 10, p. 225 (abstract section) Dec. 1937.

Schmitt and Taeger studied the variations in daily lead elimination and lead in blood in two persons with lead poisoning, their purpose being to determine fixation or mobilization of lead during various methods of treatment. In agreement with other workers, they found increased lead elimination (mobilization) associated with the use of sodium carbonate, acid diet with ammonium chloride, calcium poor diet, potassium iodide

(probably by effect on the thyroid), elytyran, and parathormone. It was remarkable that by the use of an alkaline vegetable diet and by peroral and intravenous administration of calcium, they could also induce lead mobilization. Therefore, the authors conclude that any procedure involving a shock, such as sudden diet change or sudden administration of great calcium doses, is to be avoided. (Since this contradicts the work of other investigators, and especially therapeutic experience, it seems that further tests on a wider group is necessary.--L.T.) The authors recommend calcium enrichment of the calcium impoverished organism in lead poisoning cases (the calcium impoverishment of erythrocytes is especially outstanding) by gradually increasing doses of calcium phosphate and calcium gluconate.--L. Teleky.

#### THE RELATION BETWEEN LEAD POISONING AND THE PARATHYROID GLAND.

Karl Reinhart. Arch. Gewerbepath. Gewerbehyg. 9, 80-87 (1938).  
Chemical Abstracts, vol. 33, p. 3003.

Lead poisoning affects the parathyroid's function of mineral regulation. Bone changes occur in chronic lead poisoning similar to those in cases of adenoma of the parathyroid. Serum studies upon these individuals showed hypercalcemia (Ca level, 11 mg. %) and a lowering of the inorg. P level (below 4 mg. %). These symptoms point toward hyperfunction of the parathyroid.

#### DIFFERENCE BETWEEN "CONTRACTED" KIDNEYS DUE TO LEAD AND THOSE DUE TO OTHER FORMS OF NEPHRITIS.

G. Aiello. Il Lavoro, July, 1924, vol. 15, pp. 212-214.

Abstracted in J. of Ind. Hygiene, vol. 7, no. 1, p. 12 (abstract section) Jan. 1925.

Lead nephritis presents a varying picture of both epithelial and interstitial changes and attacks either the blood vessels or the tubular epithelium. All contracted lead kidneys show in the tubules or the glomeruli inflammatory reactions which are lacking in the usual contracted kidney. When the patient has not been exposed to lead for a long time these lesions are absent. This suggests that lead nephritis is produced by the passage of lead through the excretory epithelium.--J.W.S.B.

#### SPINAL INJURIES FROM LEAD AND THE SIGNIFICANCE OF SPINAL FLUID LEAD DETERMINATION IN RECOGNIZING LEAD DAMAGE OF THE CENTRAL NERVOUS SYSTEM.

F. Duensing. Deutsch. Ztschr. f. Nervenheilk., vol. 143, pp. 297-305 (1937).

Abstracted in J. of Ind. Hygiene, vol. 19, no. 10, pp. 224-225 (abstract section) Dec. 1937.

The author describes a case with tabes-like symptoms: irregular pupils, unequal indiameter, no light reactions, no biceps, triceps or patellar reflexes, hypotonia of the musculature, sensibility decreased, Romberg +, ataxic gait, pressure in the head, and a depressive mood. Lead determination in the blood showed 72.2 gamma, that in the spinal fluid, 220 gamma, whereas in others examined it was only 18-38 gamma per 100 cc. in the spinal fluid. Thus it seems certain that a spinal lead injury is present. The cause was drinking water containing lead.

A painter who worked extensively with lead paints fell ill in 1928 with gastro-intestinal symptoms. In 1933 he suffered general weakness, headache, dizziness, attacks of unconsciousness, irritability. In 1936 these were still present along with a heavy feeling in the arms and legs. It had been many years since his last exposure. In the spinal fluid there was 493 gamma per 100 cc. and in the blood 129.5 gamma. The author mentions some cases from the old literature (Redlich's lead tabs) and notes the necessity for determining spinal fluid lead.--L. Teloky.

#### FATAL SUBACUTE OCCUPATIONAL LEAD POISONING.

W. Ehrhardt. Arch. f. Gowerbepath., vol. 9, pp. 407-413 (1939).

Abstracted in J. of Ind. Hygiene, vol. 21, no. 9, p. 212 (abstract section) Nov. 1939.

A man working with lead sprays under very bad conditions died after an illness of 4 days. He had worked only on odd occasions for some weeks. For 8 days he suffered colic, returned to work, and again fell ill. This time he had colic, vomiting, diarrhea, salivation, weakness of the muscles, transitory paresis of the legs. Necropsy showed the kidneys and liver to be damaged. The lead content of the blood was 185 u gm.% and that of the kidneys 370 u gm. in 10 gm. of dry substance.--L. Teloky.

#### DERMATITIS FROM RED LEAD.

J.A.M.A. (Queries and minor notes), vol. 112, p. 1749 (Apr. 29, 1939).

Abstracted in J. of Ind. Hygiene, vol. 21, no. 7, p. 177 (abstract section) Sept. 1939.

A man employed painting metal with red lead for 6 mos. developed a general erythematous and intensely pruritic dermatitis that covered almost the entire body. He stopped work immediately, but in spite of constant care and hospitalization, it was 1 yr. before the condition cleared up. By patch test he was found to be definitely sensitive to the paint he had been using. The reply points out that red and white lead are not widely employed in paints now; some of the pigments substituted are listed as well as the solvents. Dermatitis due to systemic lead poisoning has never been reported.--Helen Lawson.

#### OCULAR PLUMBISM IN CHILDREN.

J.L. Gibson. Brit. Jour. Ophth., Nov., 1931, vol. 15, pp. 637-642.

Abstracted in J. of Ind. Hygiene, vol. 14, no. 4, p. 86 (abstract section) Apr. 1932.

The cases dealt with in this paper have no pyrexia. The mild ones are brought by their mothers because they have developed a recent internal squint. The squint is due to paralysis of one or of both external recti. Papilledema up to 6 diopters is found, which is due to increase in intracranial tension. The severe cases are more evidently cases of lead encephalopathy. Explanation is given of why soluble lead is available, and why and how it is ingested. Lead is found in the urine. No albumin is found in the urine.

#### SHADOWS PRODUCED BY LEAD IN ROENTGEN-RAY PICTURES OF GROWING SKELETON.

E.A. Park, D. Hackson. and L. Kajdi. Abstr. as follows from Am. Jour. Dis. Child., March, 1931, vol. 41, p. 485, in Jour. Am. Med. Assn., June 6, 1931, vol. 96, p. 1989.

Abstracted in J. of Ind. Hygiene, vol. 13, no. 8, p. 201 (abstract section) Oct. 1931.

Park and associates state that, when taken for a sufficiently long period in sufficient dosage, lead apparently can produce changes in the bone in process of formation which are reflected in the roentgenograms as shadows of increased density. Like elementary phosphorus, lead can cloud bones in roentgenograms. The clouding is most conspicuous where growth is occurring most rapidly; namely, at the anterior ends of the middle six ribs, the lower ends of the femora, the upper end of the humerus, the lower ends of the radius and ulna, and at both ends of the fibula and tibia. In two cases observed by them in which the banding was most marked, only the ribs were studied with the roentgen ray; but in two other cases in which banding was well marked in the fast growing parts of the skeleton, it was not noted in the slow growing parts. That only those parts of the skeleton in process of growth at the time of ingestion of lead are affected is proved by the entirely normal structure of the old bone proximal to the lesions, the sharp limitation of the lesions to the growing parts, and a degree of involvement at any point exactly proportional to the rate of growth at that point. Obviously growth in any bone occurs in many places, but only where growth occurs rapidly do the changes readily reach such magnitude as to show in roentgenograms. The fact that only the bone in process of growth is affected must mean that the lead either enters into the chemical composition of the bone or influences cellular activity in such a way that the character of the bone formed becomes altered.

#### THE EARLY DIAGNOSIS OF ACUTE AND LATENT PLUMBISM.

F.L. Smith, 2nd, T.K. Rathmell and G.E. Marcil. Am. J. Clin. Path., vol. 8, pp. 471-508 (1938).

Abstracted in J. of Ind. Hygiene, vol. 21, no. 6, p. 142 (abstract section) June 1939.

The many symptoms that may be found in lead poisoning are listed and mention is made of the difference in susceptibility to lead. The authors review figures given as normal lead in urine and concentrations in urine of exposed and poisoned persons. In discussing the significance of stippled cells, the authors list sixteen other pathological conditions in which these cells have been reported. In the authors' series and experiments, stippling did not precede symptoms and it often was absent in the toxic stage. Reticulocyte response and basophilic aggregation are not considered adequate as diagnostic tests.

Detailed blood data are given on a large number of patients grouped according to clinical manifestations, and the authors believe that a differential diagnosis can be made by accurate determination of lead in serum, cells and fibrin fraction when considered in relation to the whole blood value. Their analytic method is given. Tests on 36 healthy persons gave as minimum and maximum whole blood values averages of 0.001 and 0.006 mg/10 gm. respectively. Corresponding values for cell and fibrin reaction were about twice those for the whole blood. Blood determinations made on 47 patients with diseases other than plumbism gave about the same averages. Climatic, seasonal and daily changes had no effect, nor did food, fatigue, violent exercise or menstruation.

It was significant that lead was never detected in the serum, but in

cases of plumbism, serum lead was present in amounts up to 0.015 gm./10 gm. In cases of chronic poisoning, lead disappears from the serum but whole blood lead values are higher than normal. If borderline cases are put on an acid diet, lead will reappear in the serum.

The long bibliography is not published but appears in the authors' reprints.--Helen Lawson.

#### CALCIUM THERAPY IN LEAD POISONING.

H. Taeger. Klin. Wehnschr., vol. 16, pp. 1613-1615 (1937).

Abstracted in J. of Ind. Hygiene, vol. 20, no. 2, p. 48 (abstract section) Feb. 1938.

The author does not favor the calcium therapy first suggested by Aub, Fairhall and their colleagues for depositing lead in the organism, and disapproves especially the recommendation of massive intravenous doses of Ca and the idea that lead poisoning can be prevented by prophylactic doses of Ca. Supported by investigations on animals and men, he comes to the view that lead is positively mobilized by any interference with the course of the disease, by dosage with potassium iodide, acids and Ca. Prevention of additional lead resorption must be achieved by preventing exposure. In severe cases the treatment must be purely symptomatic at first. Since each change of the mineral metabolism balance causes mobilization, the diet must be adjusted with the greatest exactness to the patient's ordinary diet. Each change in acidity should be offset by giving a less acid or more alkaline diet. After the first acute symptoms have passed, Ca should be given to the Ca-poor organism, but only slowly by gradually increasing amounts of milk. Then over a longer period Ca preparations in small quantities can be given. By such a procedure one can, with the greatest care, induce lead excretion and not a lead fixation. Only after a long time can the patient return to his normal diet.--L. Teloky.

#### EFFECT OF NICOTINIC ACID ON INCREASED PORPHYRINURIA OCCURRING IN SEVEN PAINTERS.

E.S. Gross, Y. Sasaki and T.D. Spies. Proc. Soc. Exp. Biol. & Med., vol. 38, pp. 289-292 (1938).

Abstracted in J. of Ind. Hygiene, vol. 21, no. 5, p. 117, (abstract section) May 1939.

Since the porphyrinuria seen in pellagra disappears after administration of nicotinic acid, the authors tried the chemical on 7 painters with at least 15 yrs. exposure to lead. As controls the urines of 45 students were used. On the first test, 13 of the 45 controls gave positive pink color, but when these 13 were again tested they were negative. It is thought that alcohol or diet may have caused the positive reactions. For 3 days the painters' urines were tested and all found strongly positive. Then for 9 days, each was given 2 daily doses (2.4 gm. altogether) of nicotinic acid. A reduction in color was seen in all the day after acid was first instituted, and at the end of 9 days the test was negative or almost so. When tests were continued for 14 days (without nicotinic acid), the color gradually returned to what it had been at the beginning.--Helen Lawson.

#### INDUSTRIAL POISONS

D. Hunter. Practitioner, vol. 137, pp. 290-313 (Sept. 1936).

Abstracted in J. of Ind. Hygiene, vol. 19, no. 4, pp. 79-80 (abstract

section) April 1937.

Of 55 patients exposed to lead hazards who attended the author's out-patient department during 5 years, 36 were judged to be suffering from toxic episodes and 19 to be free of poisoning. The exact details of his work should be obtained from the patient; his first answer often does not suggest exposure to lead or its compounds. Diagnosis of intoxication depends on the presence of one or more of these: colic, palsy, anemia and encephalopathy. Constipation and slight stippling of the red cells are insufficient, without one of the foregoing, to establish a diagnosis of poisoning. In studies of lead excretion specimens of stools and urine must be collected for at least 3 days. Since lead may be swallowed and passed unabsorbed in the feces, the only proof of absorption is to find lead in the urine. Some weeks after removal from exposure the output may read 1 mgm. per day in the feces and 0.3 mgm. in the urine. Under ammonium chloride treatment the fecal excretion may reach about 2 mgm. per day, but it is doubtful whether renal excretion ever exceeds 0.3 mgm. per day. The presence of lead in the excretion is not necessarily proof that a lead worker is suffering from poisoning, he may be insusceptible. Lead colic is ten times as common as lead palsy. The lead line round the gums is an indication of absorption only and not of intoxication. Its intensity and size provide a rough guide to the duration and severity of exposure to lead. If stippling of the red cells is detected in a sufficiently large number of microscopic fields, further exposure of the patient to lead hazards should be prevented immediately. Punctate counts are of extreme value in the prophylaxis of plumbism among lead workers, and are essential for the hygienic control of lead processes. Encephalopathy is always of serious prognostic significance. Its onset is sudden, usually with epileptiform convulsions. To-day there is grave danger of wrongly attributing to lead poisoning all symptoms occurring in persons exposed to lead. In preventive treatment the usual cleanliness precautions must be taken. A diet of high calcium content is needed, and aperients should be taken regularly. In curative treatment, too, calcium is given. When the calcium intake is sufficient most of the lead is stored in the bones, and the patient presents no symptoms. After acute toxic symptoms have passed, elimination of lead may be accelerated by low calcium diet with potassium iodide, ammonium chloride, or phosphoric acid.

In a discussion of poisoning by coal-tar derivatives it is mentioned that the position of the substituent groups in the benzene ring has a great effect on the toxic action. The death-rate in cases of benzene poisoning is high and the results of treatment poor. The use of benzene in industry should be suppressed except in entirely enclosed processes. Poisonings by chlorinated hydrocarbons and toxic gases are also briefly described.--  
T. Bedford.

#### LEAD POISONING IN INDUSTRY AND ITS PREVENTION.

M.R. Mayers and M.M. McMahon. N.Y. State Dept. Labor, Div. Indust. Hyg., Spec. Bull. 195, 1938 (Price 45 cents).

Abstracted in J. of Ind. Hygiene, vol. 20, no. 7, pp. 144-145 (abstract section) Sept. 1938.

This bulletin of 68 pages is an extremely good example of an educational effort by a State Department of Labor. In this short book, practical knowledge in relation to lead is summarized extremely well, with a

clear understanding of the difficulties and how to avoid them. Starting with the chapter on the source of lead poisoning in industry, there is then discussed the physiological effects of lead absorption and the diagnosis of lead poisoning. Then, there is a long chapter on the prevention of lead poisoning in various industries, followed by the relationship of the problem to the law. All of the writing in the book is well done, and the problems appear to be fairly and accurately stated. It is sufficiently simple so that nearly anyone should be able to understand it and, therefore, brings to the layman as well as the physician an understanding of the problem of lead poisoning in a simple and accurate way. Lead poisoning is one of the most difficult problems in industrial disease, because it is so difficult to diagnose borderline cases. This book satisfactorily brings out the distinction between lead absorption and lead poisoning.

The pamphlet appears so well done that the reviewer hesitates to refer to two minor criticisms: First, the treatment of acute lead episodes is not mentioned. In this condition the use of calcium both by mouth and intravenously is so satisfactory that it is interesting that mention of this was neglected. Secondly, though there are many illustrations, some are not very clear and do not impress one as always representing the best arrangements for avoidance of lead hazards. This is particularly true in regard to the respirators worn by workers.

On the whole, this special bulletin deserves very great praise. It should be of value to industries and industrial commissions throughout the United States.--J.C. Aub.

#### THE PREVALENCE AND DISTRIBUTION OF INDUSTRIAL LEAD POISONING.

A. Hamilton. Jour. Am. Med. Assn., Aug. 23, 1924, vol. 83, pp. 583-588.

Abstracted in J. of Ind. Hygiene, vol. 7, no. 1, p. 10 (abstract section) Jan. 1925.

Industrial conditions in the lead trades have been greatly improved in the last fourteen years, but there is room for further improvement in some plants. The actual prevalence of the disease is not known because cases are not reported.

Industries with a lead hazard are discussed, particularly the painters' and printers' trades. The forms of plumbism differ in different industries and are partly dependant on the severity of the exposure. In twelve years 131 cases of lead encephalopathy have come to the author's notice, and all of these followed exposure to lead dust or fumes. The effective prevention of lead poisoning lies in purification of the air which the workman breathes rather than in correction of his personal habits. The disease is far more important than many of our common infectious diseases, and it deserves far more interest devoted to its detection, diagnosis, and prevention.--J.C.A.

#### THE LEAD WORKER'S FAMILY.

L. Devoto. Il Lavoro, Sept., 1924, vol. 15, pp. 259-261.

Abstracted in J. of Ind. Hygiene, vol. 7, no. 1, p. 14 (abstract section) Jan. 1925.

In an address to the first Eugenic Congress at Milan, the author gave his opinion that the lead worker's family is numerically and physically

inferior and has a disposition to occupational and other diseases unless special measures are taken. He recommends employment only of young men of good health and good heredity; instruction of workers as to the premonitory signs of lead poisoning (presaturnism); periodic examinations; and sanitary measures.--J.W.S.B.

LEAD POISONING. II. 51 FATAL CASES OF SUSPECTED LEAD POISONING; AND 46 OF NON-FATAL LEAD POISONING; DIAGNOSIS; TREATMENT; SUMMARY.

V.A. Gant. Ind. Med., vol. 7, pp. 679-99 (November 1938).

Abstracted in J. of Ind. Hygiene, vol. 21, no. 4, p. 94 (abstract section) April 1939.

The author devotes considerable space to case histories of patients classified as lead poisoning among children, as chronic industrial cases, as doubtful and unjustified cases, and as acute cases, with discussions in each instance. In the concluding sections of the paper, the author repeats that since there are no symptoms pathognomonic of lead alone, diagnosis without chemical findings (determination of lead in blood or urine) is incomplete. Disodium phosphate is proposed as the best therapeutic agent in the treatment and should be used in conjunction with a normal calcium diet.--W.H. Buck.

ATMOSPHERIC CONTENT OF LEAD AND ITS CORRELATION WITH BASOPHILIC AGGREGATION TESTS IN EXPOSED STORAGE BATTERY WORKERS.

C.P. McCord, H.T. Walworth, J. Johnston and P.E. Fisher. Indust. Med., vol. 6, pp. 357-363 (June 1937).

Abstracted in J. of Ind. Hygiene, vol. 19, no. 9, p. 195 (abstract section) Nov. 1937.

Dr. McCord and his colleagues have carried on their survey of lead workers to include employees of 13 small storage battery plants in half of which only 2 men were employed. Since it is possible to breach such a business with as little as \$600, it is not surprising to find these plants operating under primitive conditions, as far as safety measures are concerned. The result is a good deal of lead poisoning, even though there is an enormous labor turnover in such plants.

Lead determinations in the air of 8 plants are listed along with the corresponding basophilic aggregation counts (in percentages of the total red blood count). The count for non-exposed persons is about 1.5. In tests made on 400 employees of a large plant, nine men had counts above normal; these men were found to work at identical jobs within a radius of 30 ft. Tests of atmospheric lead in this area showed it to be the only one in the plant where the concentration was high enough to cause trouble. "In these plants leading to quantitative determinations of lead in the atmosphere in excess of the threshold of tolerability, corresponding basophilic aggregation tests were likewise such as to fall beyond the zone characterizing healthy, unexposed persons." Suggestions for standardizing the aggregation test are included.--Helen Lawson.

LEAD STEARATE POISONING IN THE RUBBER INDUSTRY.

H.J. Cronin. Boston Med. and Surg. Jour., May 7, 1925, vol. 192, p. 900.

Abstracted in J. of Ind. Hygiene, vol. 7, no. 10, pp. 171-172 (abstract section) Oct. 1925.

Nine employees in a rubber factory were affected by an unusual form of lead poisoning caused by lead oxide or litharge in loose chemical combination with stearic acid and sulphur. Stearic acid ( $C_{18}H_{36}O_2$ ) is associated with palmitic and oleic acids as a mixed ether in solid animal fats or tallow. It was used in the rubber industry to soften the rubber. The heat of the mill turned solid stearic acid into an oily liquid that flowed over the mixer's hands and arms which were also covered with lead oxide powder.

A black deposit of lead stearate on the exposed parts of the hands, arms, and face was the characteristic symptom. There was slight itching followed by a subacute dermatitis with dryness of the skin, fissures, and desquamation. There were no constitutional symptoms of lead poisoning as this method of using stearic acid was discontinued and the men were put on other employment temporarily. A protective ointment was applied with loose bandages, and after five days the dermatitis subsided.

The author suggests that if stearic acid must be used with lead oxide the workmen should have their arms covered and should wear gloves. On the other hand, there is danger that long sleeves and gloves may get caught and draw the worker's arm between the rollers. Even if the acid is shoveled on to the mill it is often necessary to handle small pieces that drop and to handle the mixed rubber when it is cut from the mill. Scrupulous cleanliness should be observed. It is possible for rubber chemists to combine stearic acid with other solvents in such a way as to prevent the reaction with lead on the workers' arms.

#### LEAD IN THE PRINTING INDUSTRY.

John M. Hepler, Paul F. Rezin and R.W. Colina.  
J. Ind. Hyg. Toxicol. 20, 641-5 (1938).  
Chemical Abstracts, vol. 33, p. 2608.

Air samples from 14 plants were obtained by the impinger method. Operators were found to be breathing air contg. as high as 3.8 mg. of lead per 10 cu.m. of air. Lead pots are health hazards.

#### LEAD HAZARD AMONG AUTOMOBILE BODY WORKERS AND ITS PREVENTION.

E. Truby. Arbeitsschutz, pp. 253-254 (1938).

Abstracted in J. of Ind. Hygiene, vol. 21, no. 3, p. 71 (abstract section) Mar. 1939.

Lead poisoning occurred frequently among men soldering sheet metal and joints in an automobile body factory. The solder contains 75% lead. After it hardens, it is polished by hand or by power driven wheels. Hand filing produces coarse dust, the machine grinding produces a dangerous fine dust. The danger should be avoided by replacing the solder by a coat of cement-like material. The solder should be heated only until it has a paste-like consistency, not to the point where it becomes volatile, and grinding wheels should not be used. Cleanliness and medical supervision are important.--L. Teleky.

#### LEAD HAZARD AT SOLDERING BENCHES.

T. Hatch. N.Y. State Dept. Labor, Indust. Bull., vol. 18, p. 284 (June, 1939).

Abstracted in J. of Ind. Hygiene, vol. 21, no. 8, p. 200 (abstract

section) Oct. 1939.

In this brief article, the author considers the lead hazard arising from the lead fumes of soldering and the dust and cuttings released by filing, sanding and grinding. A drawing of the recommended bench is included; this bench is provided with a ventilated grilled top and a lateral exhaust hood. The rate of ventilation is based on an average velocity of 75 c.f.m. through the table top. This method will not control lead dust given off by a high speed grinder.--Leslie Silverman.

#### OCCUPATIONAL HEALTH HAZARDS IN MASSACHUSETTS INDUSTRIES. II. PAINT AND VARNISH MANUFACTURE.

Mass. State Dept. Labor and Indust., Div. Occupat. Hyg., Boston, (January, 1938).

Abstracted in J. of Ind. Hygiene, vol. 20, no. 7, p. 145 (abstract section) Sept. 1938.

The chemical health hazards in the paint manufacturing industry were studied in 8 establishments, and the most important one was found to be lead.

The lead exposure of workers in the departments chiefly affected was determined in seven of the plants. It was found that when lead paint is being mixed, the average amount breathed by workers in the mixing department is about  $2\frac{1}{2}$  mgm. of lead per day. This is 1 mgm. greater than the permissible value of one and a half milligrams.

The precautions taken in this industry were described and critically discussed. Conditions on the average would seem conducive to occasional mild cases of lead poisoning; and when the customary precautions are omitted more severe cases would seem possible.--Authors' Summary.

#### HYGIENIC METHODS OF PAINTING: THE DAMP RUBBING DOWN PROCESS.

Jour. Roy. Soc. Arts., Feb., 1923, vol. 71, pp. 240-255.

Abstracted in J. of Ind. Hygiene, vol. 5, no. 4, pp. 81-82 (abstract section) Aug. 1923.

This is a very interesting article which deals with the importance of dry sandpapering in the production of lead poisoning among painters. Figures are given showing the amount of lead dust and its distribution when walls are sandpapered. The dust may be inhaled not only during the actual process, but later from the dirty clothing. Sandpapering on wet days results in but little dust. The dust from this process contains particles of silicon, which may explain the high rate of respiratory disease in painters.

All of the dangers from this process may be obviated by moistening the surface of the wall and using a waterproof sandpaper. This is now manufactured successfully in America, and gives very satisfactory results without the production of any deleterious dust. The abandonment of dry processes for rubbing down is very desirable in respect to all paints.--J.C.A.

#### LEAD POISONING IN CIGAR MAKERS.

G.H.W. Jordans, A. Zijlmans, and J. Broos.

Nederl. Tijdschr. v. Geneesk., vol. 80, pp. 304-311 (1936).

Abstracted in J. of Ind. Hygiene, vol. 18, no. 8, p. 129 (abstract section) Oct. 1936.

Proof is furnished that the chronic ill health from which these cigar makers suffered was due to lead poisoning. They had a long and very similar history of periodic attacks of severe abdominal pain, nausea, vomiting and obstinate constipation. The abdominal wall was hard and indrawn while the urine was dark. Such possibilities were first thought of as intestinal obstruction, appendicitis and renal colic. The temperature, pulse and sedimentation rate, however, were normal. There remained the possibility of a paroxysmal porphyrinuria but the urine was not dark enough and other symptoms were absent. It was the blood examination which gave the clue to the diagnosis with its demonstration of punctate basophilia or a reticulocytosis. The pallid appearance of the patient and the delicate colored line upon the gums made it certain that the disease was one of chronic lead poisoning. One or two of the patients suffered from paresis of the extensors of the wrist. A high degree of porphyrinuria was a significant symptom and is considered to be very useful for early diagnosis of the condition. This symptom was particularly interesting in one of the patients, who suffered from a distinct hypersensitiveness to light. No sign of deposition of lead was found by x-ray examination at the ends of the bones, such as has been found in children. With the establishment of the cause of the condition came the question of the source of the lead. Drinking water was excluded. The zinc plates used by the cigar makers were found to contain as much as 1% of lead and there was no difficulty in tracing the transference of material from this plate to the mouth during use. As wooden plates would serve equally well for the manipulations involved in cigar making, it seems obvious that legislation is required to make their use obligatory.--Bull. Hyg.

#### NEW SOURCE OF LEAD POISONING.

Mornac. Ann. d'hyg., pp. 362-364 (1938).

Abstracted in J. of Ind. Hygiene, vol. 21, no. 2, p. 44 (abstract section) Feb. 1939.

The author describes the use of great quantities of lead arsenic in insecticides used by farmers and the complete carelessness in handling of it. He saw lead poisoning cases and thus recommends the use of aluminum arsenates.--L. Teleky.

#### LEAD-POISONING CAUSED BY HANDLING LEAD LINING TEA-CHESTS.

G.A. Stephens. Abstr. as follows from Med. Officer, 1931, vol. 46, p. 78, in Bull. Hyg., Nov., 1931, vol. 6, pp. 841-842.

Abstracted in J. of Ind. Hygiene, vol. 14, no. 4, pp. 85-86 (abstract section) April 1932.

This is an interesting case of lead palsy arising in a man aged 40, who had been employed in a grocery store for twenty-six years, with a brief interval of two and one-half years when he served in the war. He had wrist drop (right) and foot drop (left) and a systolic blood pressure of 200; pyorrhea was very bad and no blue line could be seen. Part of his duties consisted in emptying tea out of lead-lined boxes, collecting the lead, and packing it for carriage by rail; and there was no doubt that the poison had been absorbed from the lead dust on his hands.

Two points of special interest arise: first, that in spite of repeated facilities for absorption nearly twenty-six years elapsed before symptoms appeared; second, that if lead oxide does form in these linings, does it remain exposed for a length of time sufficient to form so much dust that, mixing with the tea, it might give rise to lead poisoning among tea drinkers? --H.H.S.

#### EXAMPLE OF AN INSTALLATION FOR THE CONTROL OF THE HYGIENIC EXPOSURE ARISING FROM THE APPLICATION OF POTTERY GLAZES.

S.C. Rothmann. Ind. Mod., vol. 8, pp. 8-12 (Jan. 1939).

Abstracted in J. of Ind. Hygiene, vol. 21, no. 5, p. 125 (abstract section) May 1939.

This paper covers an environmental survey of a pottery plant where "dinnerware" is manufactured. The lead concentration in the plant ranges from an average of 1.2 mg per 10 cu.m. to 167 mg per 10 cu.m. With an air velocity of 400 f.p.m. in the face of the spray booth, the lead concentration near the spray machine operator varied from 0.7 to 65.0 mg per 10 cu.m., the average being 4.4 mg.

The allowable concentration of lead in air suggested by the U.S. Public Health Service is 1.5 mg per 10 cu.m.--George M. Reece.

#### TETRA-ETHYL LEAD POISONING. CLINICAL ANALYSIS OF A SERIES OF NONFATAL CASES.

R.A. Kehoe. Jour. Am. Med. Assn., July 11, 1925, vol. 85, pp. 108-110.

Abstracted in J. of Ind. Hygiene, vol. 7, no. 12, p. 208 (abstract section) Dec. 1925.

In man, poisoning by tetra-ethyl lead is produced as a result of inhalation of the vapor and as a result of skin absorption. Subjective symptoms are: an early and troublesome insomnia; nausea, anorexia, and vomiting, especially in the early morning; vertigo and dull headache; and muscular weakness. Objective signs are: Pallor, subnormal blood pressure, subnormal temperature, loss of weight, tremor, occasional lead line, and excretion of lead in urine and feces.

In severe cases, a central nervous system involvement is the essential characteristic. The patient is in a constant state of excitement which may progress to mania and death from exhaustion.

Kehoe's treatment, which gave good therapeutic results, consisted in the daily administration of from 20 to 30 gm. of mixtures of sodium bicarbonate or sodium citrate, magnesium oxide, and calcium carbonate. In more severe cases magnesium sulphate was added. No narcotics were used.--K.R.D.

#### THE PHYSIOLOGICAL EFFECTS OF SMALL AMOUNTS OF LEAD: AN EVALUATION OF THE LEAD HAZARD OF THE AVERAGE INDIVIDUAL.

A.S. Minot. Physiological Reviews, vol. 18, pp. 554-577 (Oct. 1938).

Abstracted in J. of Ind. Hygiene, vol. 21, no. 7, p. 165 (abstract section) Sept. 1939.

This is an excellent summary of recent investigations in regard to lead poisoning. It covers briefly most of the important problems related

to the subject, such as distribution of lead, a survey of normal lead exposures, sources of "normal" lead, and the like. There is then a discussion of the amount of lead causing poisoning, and the methods of absorption. There is also a discussion of the biochemical and chemical action of lead on the body. The article is practically entirely a summary of the existing literature and as such is extremely well written, with the important references and, on the whole, a good evaluation of divergent points of view. As it is written by an investigator who has contributed valuable material to the problem, it is a very convenient way to become readily and authoritatively conversant with the present knowledge.--Joseph C. Aub.

#### LEAD POISONING, WITH SPECIAL REFERENCE TO POISONING FROM LEAD COSMETICS.

M. Barron and H.C. Habein. Abstracted as follows from Am. Jour. Med. Sc., Dec. 1921, 162, No. 6, 833, in Jour. Am. Med. Assn., Feb. 4, 1922, 78, No. 5, 362.

Abstracted in J. of Ind. Hygiene, vol. 4, no. 5, p. 68 (abstract section) Sept. 1922.

"In the cases reported by Barron and Habein a powder containing pure lead carbonate, ground to an impalpable powder used as a face powder, was responsible for the lead poisoning. It is urged that rigid laws be enacted prohibiting the sale of any compound containing lead for cosmetic purposes."

#### A TYPICAL LEAD POISONING CAUSED BY TAP WATER.

O. Altmann and K. Nowotny. Wien. klin. Wchnschr. vol. 49, pp. 613-616 (1936).

Abstracted in J. of Ind. Hygiene, vol. 18, no. 8, p. 129 (abstract section) Oct. 1936.

In July, 1932, the appendix was removed from a woman. She had further complaints of anemia and radial and shoulder paralysis which gradually developed up to April, 1934.

A man who had first an appendectomy had a continuation of his complaints and radial and shoulder paralysis.

In two other persons complete hand and shoulder paralysis, digestive disturbances and anemia were found. In the drinking water of the city in question there were found 5.5 mg. of lead per liter of water.--L. Teloky.

#### MEDICOLEGAL ASPECTS OF DISABILITY IN INDUSTRIAL LEAD POISONING.

M. Kummel. New Jersey Med. Soc. Jour., April, 1931.

Abstracted in J. of Ind. Hygiene, vol. 14, no. 3, p. 79 (abstract section) March 1932.

The author cites twelve cases of physical disability following lead poisoning, and summarizes his findings as follows:

1. A mild case of lead poisoning may leave no permanent disability.
2. The gastro-intestinal type of lead poisoning is of a temporary nature and the resulting persistent constipation is not disabling in character.

3. The cerebrospinal form of lead poisoning is the most distressing in its manifestations, most destructive in nature, most permanent in character, and may result in total permanent disability.

4. The kidneys usually show the degenerative changes.

5. Long hours and overwork are conducive to greater disability.

6. Chronic lead poisoning sufferers have a lowered resistance, are poor risks for health insurance, and have a shorter life expectancy.--L.T.F.

#### LEAD POISONING LEGISLATIONS AND STATISTICS.

F.L. Hoffman. Prudential Insurance Co. of America, Newark, N.J., pp. 40, 1933.

Abstracted in J. of Ind. Hygiene, vol. 16, no. 1, pp. 3-4 (abstract section) Jan. 1934.

Hoffman discusses the progress of legislation prohibiting or limiting the use of lead paint. A Convention to prohibit it, with some exceptions, was adopted by the International Office in 1921 but the only important industrial country which has adhered to this Convention so far is France, although the 18 others include Czechoslovakia, Austria, Belgium, Poland, Spain, and Sweden. England has stiffened the regulations designed to protect users of lead paint and is able to show a distinct falling off in the number of certified cases in 1931 as compared with the three previous years, an improvement attributed by the authorities in part to the new regulations, although they admit that lack of employment also plays a part. (For other lead trades the English statistics include the numbers employed, but it has never been possible to ascertain the number of painters employed in any one year.) Germany also regulates instead of prohibiting the use of lead paint and there also the fall in the number of cases is attributed in part to these protective measures, which include forbidding dry rubbing, regulating spray painting, providing means for the determination of lead in paint, forbidding the work of women and of men under 18 years, insisting on washing facilities, special working clothes, and a semi-annual medical examination. In the countries where lead paint is prohibited no improvement can be shown, even though several of them adopted the law several years ago, because none of them has statistical data of any value.

Hoffman analyses the deaths in the Registration Area of the United States which numbered 134 in 1929, 101 in 1930 and 111 in 1931. This last figure includes 8 children, and omitting them the average age at death is found to be 53.7 years. Painters made up 55 of the 103 adults, dying at an average age of 55.6 years. The quantity of lead consumed in industry was 972,000 tons in 1929, 567,000 in 1931.

The death rates from lead poisoning during the years from 1920 to 1931 are given, but they are calculated on the number of the population, which means little. Only the English statistics, which give the number employed in the various industries each year, show whether there has been a real increase or decrease in lead poisoning. In this country no such data are available and the fact that the rate was 1.2 in 1928 and only 0.85 in 1930 is of no real significance.

Brief clinical reports are given of most of the 101 fatal cases in 1930 and the prevalence of chronic nephritis, arteriosclerosis, cerebral hemorrhage, chronic myocarditis, as complications, strikes the eye, but without comparable material from a control group of non-lead workers it is impossible to say if it means anything. In some cases, as Hoffman says, the diagnosis is far from convincing, e.g., a printer dying of aplastic anemia complicated by myocardial insufficiency.

The occupational disease statistics for 1931 in New York State are given, showing 99 cases of lead poisoning, of which, however, 18 were disallowed. The painting trade led with 31 cases (6 of them disallowed), then came storage battery manufacture with 26 (two disallowed), and then lead arsenate (manufacture or use?) with 10. No other industry had as many as five allowed cases.

There is a discussion of lead poisoning in the U.S. Navy Yards and the regulations which have been adopted to prevent such cases in the future; there is an analysis of the deaths in the Registration Area during the years 1925-1930 and the chief complications, and finally the translation from a Swiss report on the question of the prohibition of white lead paint.--Alice Hamilton.

## SELECTED REFERENCES

Aliavdin, N., and Peregood, E.: Lead Content in the Duodenal Juice in Cases of Saturnism. Preliminary Report. Jour. Ind. Hyg., vol. 18, no. 3, p. 139, March 1936.

Aub, Joseph C., Fairhall, Lawrence T., Minot, A.S., and Reznikoff, Paul: Lead Poisoning. Jour. Ind. Hyg., vol. 7, no. 11, pp. 531-537, Nov. 1925.

Aub, J.C., Minot, A.S., Fairhall, L.T., and Reznikoff, P.: Recent Investigations of Absorption and Excretion of Lead in the Organism. Jour. Am. Med. Assn., vol. 83, pp. 588-591, Aug. 23, 1924.

Barnes, E.C.: Possibilities of Control of Lead Exposure by Examining less than 24 Hour Urine Samples. Jour. Ind. Hyg., vol. 21, no. 9, pp. 464-468, Nov. 1939.

Belknap, Elston L.: Clinical Studies on Lead Absorption in the Human. III. Blood Pressure Observations. Jour. Ind. Hyg., vol. 18, no. 7, pp. 380-390, Sept. 1936.

Bloomfield, J.J., and Isbell, H.S.: The Presence of Lead Dust and Fumes in the Air of Streets, Automobile Repair Shops, and Industrial Establishments of Large Cities. Jour. Ind. Hyg., vol. 15, no. 3, pp. 144-149, May 1933.

Blumgart, Herrmann L., M.D.: Lead Studies: VI. Absorption of Lead by the Upper Respiratory Passages. Jour. Ind. Hyg., vol. 5, no. 5, pp. 153-158, Sept. 1923.

Brown, Lieutenant Commander, E.W., (M.C.), U.S.N.: A Study of Lead Poisoning Among Oxyacetylene Welders in the Scrapping of Naval Vessels. Jour. Ind. Hyg., vol. 8, no. 3, pp. 113-140, March 1926.

Clifford, P.A.: Report of (The Determination of) Lead (in Foods). Jour. Assoc. Agr. Chem., vol. 21, pp. 212-218, 1938.

Craig, D. Norman, and Vinal, George W.: Solubility of Lead Sulfate in Solutions of Sulfuric Acid, Determined by Dithizone with a Photronic Cell. Jour. Research Natl. Standards 22, pp. 55-70, 1939, (Research Paper No. 1165).

Crawford, B.L., Stewart, H.L., Willoughby, C.E., and Smith, F.L.: Distribution of Lead in the Cat after Intravenous Injection of a Colloidal Lead Preparation, and the Effect of Irradiation on This Distribution. Am. Jour. Cancer, vol. 33, pp. 401-422, 1938.

Davis, C.M., M.D.: Lead Poisoning in a Golf Professional: Case Report. Jour. Ind. Hyg., vol. 5, no. 7, pp. 253-254, Nov. 1923.

Dean, Archibald S., M.D., Dr. P.H.: An Epidemic of Lead Poisoning Caused by the Sandpapering of Automobile Bodies. Jour. Ind. Hyg., vol. 6, no. 6, pp. 245-250, Oct. 1924.

Dolowitz, D., Fazekas, J.F., and Himwich, H.E.: The Effect of Lead on Tissue Metabolism. Jour. Ind. Hyg., vol. 19, no. 2, pp. 93-94, Feb. 1937.

Fairhall, Lawrence T., A.M., Ph.D., and Shaw, Charlotte P., B.S.: The Deposition of Lead Salts, with a Note on the Solubilities of Di-Lead Phosphate in Water at 25°C. and of Di-Lead and Tri-Lead Phosphates in Lactic Acid at 25°C. Jour. Ind. Hyg., vol. 6, no. 4, pp. 159-168, Aug. 1924.

Fairhall, Lawrence T.: The Lead Content of Evaporated Milk. Jour. Ind. Hyg., vol. 19, no. 9, pp. 491-497, Nov. 1937.

Fairhall, Lawrence T.: Note on the Accuracy of Lead Analyses. Jour. Ind. Hyg., vol. 15, no. 5, pp. 289, Sept. 1933.

Fairhall, Lawrence T.: The Solubility of Various Lead Compounds in Blood Serum. Jour. Biol. Chem., vol. 60, pp. 481-484, July 1924.

Fine, J.: The Phagocytosis of Lead Compounds and Their Influence on the Activity of the Leukocyte. Jour. Ind. Hyg., vol. 5, no. 4, pp. 138-144, Aug. 1923.

Flinn, Frederick B., and Smith, Adelaide Ross: The Effect of Viosterol on the Excretion of Lead. Jour. Ind. Hyg., vol. 15, no. 3, pp. 156-159, May 1933.

Grant, R. Lorimer, Calvary, Horbert O., Laug, Edwin P., and Morris Herman J.: The Influence of Calcium and Phosphates on the Storage and Toxicity of Lead and Arsenic. Jour. Pharmacol., vol. 64, pp. 446-457, 1938.

Hamilton, Alice, M.D.: Lead Poisoning in American Industry. Jour. Ind. Hyg., vol. 1, no. 1, pp. 8-21, May 1919.

Hamilton, A.: Indust. Toxicology. Harper and Brothers, New York, 1934, pp. 20-63.

Hanzlik, P.J., and Presko, E.: Comparative Toxicity of Inorganic Lead Compounds and Metallic Lead for Pigeons. Jour. Pharmacol. and Exper. Therap., vol. 21, pp. 123-129, March 1923.

Hanzlik, P.J., and Presko, E.: Comparative Toxicity of Metallic Lead and Other Heavy Metals for Pigeons. Jour. Pharmacol. and Exper. Therap., vol. 21, pp. 145-150, March 1923.

Hanzlik, P.J., and Presko, E.: Therapeutic Efficiency of Various Agents for Chronic Poisoning by Metallic Lead in Pigeons. Jour. Pharmacol. and Exper. Therap., vol. 21, pp. 131-143, March 1923.

Harrold, G.C., Mook, S.F., and Holden, F.R.: A Practical Method for the Rapid Determination of Lead When Found in the Atmosphere. Jour. Ind. Hyg., vol. 18, no. 10, pp. 724-732, Dec. 1936.

Kehoe, Robert A., Thamann, Frederick, and Cholak, Jacob: An Appraisal of the Lead Hazards Associated with the Distribution and Use of Gasoline Containing Tetraethyl Lead. Part I. Jour. Ind. Hyg., vol. 16, no. 2, pp. 100-128, March 1934.

Kehoe, Robert A., Thamann, Frederick, and Cholak, Jacob: An Appraisal of the Lead Hazards Associated with the Distribution and Use of Gasoline Containing Tetraethyl Lead. II. The Occupational Lead Exposure of Filling Station Attendants and Garage Mechanics. Jour. Ind. Hyg., vol. 18, no. 1, pp. 42-68, Jan. 1936.

Kehoe, Robert A., Thamann, Frederick, and Cholak, Jacob: Lead Absorption and Excretion in Certain Lead Trades. Jour. Ind. Hyg., vol. 15, no. 5, pp. 306-319, Sept. 1933.

Kehoe, Robert A., Thamann, Frederick, and Cholak, Jacob: Lead Absorption and Excretion in Relation to the Diagnosis of Lead Poisoning. Jour. Ind. Hyg., vol. 15, no. 5, pp. 320-340, Sept. 1933.

Kehoe, Robert A., Thamann, Frederick, and Cholak, Jacob: On the Normal Absorption and Excretion of Lead. I. Lead Absorption and Excretion in Primitive Life. Jour. Ind. Hyg., vol. 15, no. 5, pp. 257-272, Sept. 1933.

Kehoe, Robert A., Thamann, Frederick, and Cholak, Jacob: On the Normal Absorption and Excretion of Lead. II. Lead Absorption and Lead Excretion in Modern American Life. Jour. Ind. Hyg., vol. 15, no. 5, pp. 273-288, Sept. 1933.

Kehoe, Robert A., Thamann, Frederick, and Cholak, Jacob: On the Normal Absorption and Excretion of Lead. III. The Sources of Normal Lead Absorption. Jour. Ind. Hyg., vol. 15, no. 5, pp. 290-300, Sept. 1933.

Kehoe, Robert A., Thamann, Frederick, and Cholak, Jacob: On the Normal Absorption and Excretion of Lead. IV. Lead Absorption and Excretion in Infants and Children. Jour. Ind. Hyg., vol. 15, no. 5, pp. 301-305, Sept. 1933.

Key, J.A.: Lead Studies. IV. Blood Changes in Lead Poisoning in Rabbits; Stippled Cells. Am. Jour. Physiol., vol. 70, p. 86, Sept. 1924.

Klein, C.A.: Constructive Industrial Hygiene in the India Rubber Industry. Jour. Soc. Chem. Indust., vol. 41, no. 15, pp. 325R-328R, Aug. 15, 1922.

Klein, C.A.: The Prevention of Lead Poisoning in Industry. Part I. The India Rubber Industry. (Methods of Concentration of Controlled Risk). Jour. Ind. Hyg., vol. 8, no. 7, pp. 296-299, July 1926.

Kober, G., and Hayhurst, E.: Industrial Health. P. Blakiston's Son and Co., 1924, pp. 412-470.

Kogan, Dr. B., and Smirnowa, Dr. L.: Changes in the Blood Picture Under the Influence of Lead, and Their Importance in the Differential Diagnosis. Jour. Ind. Hyg., vol. 9, no. 10, pp. 435-452, Oct. 1927.

Krafka, Joseph Jr., M.D.: The Effect of Repeated Leading on the Blood Picture in Guinea-Pigs. Jour. Ind. Hyg., vol. 17, no. 1, pp. 13-17, Jan. 1935.

Krans, Edward W., and Ficklen, J.B.: A Colorimetric Method for the Detection and Estimation of Small Amounts of Lead. Jour. Ind. Hyg., vol. 13, no. 4, pp. 140-143, April 1931.

Lane, Ronald E., M.B. (Lond.), M.R.C.P. (Lond.), and Lewy, F.H., M.D. (Philadelphia, Pa.): Blood and Chronaximetric Examination of Lead Workers Subjected to Different Degrees of Exposure: A Comparative Study. Jour. Ind. Hyg., vol. 17, no. 3, pp. 79-92, May 1935.

Lane, Ronald E.: Lead Burning: A Report Upon an "Exhausted Blow Pipe." Jour. Ind. Hyg., vol. 18, no. 7, pp. 391-400, Sept. 1936.

Lane, Ronald E.: The Prevention of Industrial Plumbism. Lancet, pp. 206-211, July 25, 1936.

Lane, Ronald E., M.B., B.S. (Lond.), M.R.C.P. (Lond.): The Role of Punctate Basophilia in the Control of Industrial Plumbism. Jour. Ind. Hyg., vol. 13, no. 8, pp. 276-284, Oct. 1931.

Leake, J.P.: Text of Full Report of Investigation of Health Hazards from Tetra-Ethyl Lead Gasoline. Treasury Department, U.S. Pub. Health, Ser., pp. 61- and 57 tables, 1926, (bound separately).

Lewy, F.H., M.D.: The Application of Chronaximetric Measurement to Industrial Hygiene Particularly to the Examination of Lead Workers. Jour. Ind. Hyg., vol. 17, no. 3, pp. 73-78, May 1935.

McCord, C.P., Minster, D.K., and Rohm, E.: The Basophilic Aggregation Test in Lead Poisoning. Jour. Am. Med. Assn., vol. 82, pp. 1759-1763, May 31, 1924.

McKail, David, M.D., D.P.H.: Prophylaxis in Industrial Lead Poisoning. Jour. Ind. Hyg., vol. 8, no. 2, pp. 74-77, Feb. 1926.

McNair, L.C., and Price, C.W.: The Effect of Rubbing Down and Scraping by Dry Process of Lead Painted Surfaces of Iron and Steel Structures. Jour. Ind. Hyg., vol. 11, no. 6, pp. 175-181, June 1929.

McNally, W.: Toxicology. Industrial Medicine, Publishers, Chicago, 1937, pp. 147-198.

Makaritschewa, Anna and Glagolewa, Tatiana: Ulcers of the Stomach in Lead Workers. Jour. Ind. Hyg., vol. 16, no. 4, pp. 201-202, July 1934.

Mayers, May R., M.A., M.D.: Lead Absorption and Compensation. Jour. Ind. Hyg., vol. 11, no. 4, pp. 124-138, April 1929.

Mayers, May R., M.A., M.D.: Lead Anemia. Jour. Ind. Hyg., vol. 8, no. 5, pp. 222-231, May 1926.

Mayers, May R., M.A., M.D.: A Study of the Lead Line, Arteriosclerosis,

and Hypertension in 381 Lead Workers. Jour. Ind. Hyg., vol. 9, no. 6, pp. 239-250, June 1927.

Minot, A.S., Ph.D.: Lead Studies 5. A. The Distribution of Lead in the Organism after Absorption by the Gastro-Intestinal Tract. Jour. Ind. Hyg., vol. 6, no. 4, pp. 125-136, Aug. 1924.

Minot, A.S., Ph.D.: Lead Studies 5. B. The Distribution of Lead in the Organism after Absorption by the Lungs and Subcutaneous Tissue. Jour. Ind. Hyg., vol. 6, no. 4, pp. 137-148, Aug. 1924.

Minot, A.S., Ph.D., and Aub, J.C., M.D.: Lead Studies 5. C. The Distribution of Lead in the Human Organism. Jour. Ind. Hyg., vol. 6, no. 4, pp. 149-158, Aug. 1924.

Moliter, P., Arnoldson, M., and Hausser, G.: Detection of Lead Poisoning in a Motor Car Factory. Arch. Mal. Profes., vol. 1, pp. 124-128, 1938.

Newman, Bernard J., McConnell, William J., Spencer, Octavius M., and Phillips, Frank M.: Lead Poisoning in The Pottery Trades. U.S. Public Health Service, Public Health Bulletin No. 116, May, 1921. Pp. 220 with index. Washington: Government Printing Office, 1921.

Norris, C., and Gettler, A.O.: Poisoning by Tetra-Ethyl Lead: Post-mortem and Chemical Findings. Jour. Am. Med. Assn., vol. 85, pp. 818-820, Sept. 12, 1925.

Nye, L.J. Jarvis, M.B., Ch. M.: Chronic Nephritis and Lead Poisoning. Pp. 138 and bibliography. Sydney: Angus & Robertson, Ltd., 1933.

Occupation and Health. International Labour Office, Geneva, vol. 2, pp. 99-141.

Oliver, Thomas: Saturnine Asthma: Is There Such a Malady? Lancet, vol. 203, No. 5174, pp. 907-909, Oct. 28, 1922.

Otto, H., and Kuhlman, F.: Gastro-Intestinal Tract, Especially Disturbance of the Intestines, in Lead Poisoning. Klin. Wchnschr., vol. 18, pp. 1081-1084, 1939.

Painters' Colic: How Caused and How Best Prevented. Reprinted from Home Office Form 394. H.M. Stationery Office, June 1923, pp. 2.

Pedley, Frank G., M.D., Dr. P.H.: The Effects of Lead on the Vision: A Case of Subhyaloid Hemorrhage. Jour. Ind. Hyg., vol. 12, no. 10, pp. 359-363, Dec. 1930.

Perlman, J.L.: Rapid Colorimetric Determination of Lead in Maple Sirup. Ind. Eng. Chem., Anal. Ed. 10, pp. 134-135, 1938.

Rezin, Paul F., and Drinker, Philip: Volatilization of Lead Below 800°C. Jour. Ind. Hyg., vol. 21, no. 9, pp. 461-463, Nov. 1939.

Sautter, Albert C.: Encephalopathy with Ocular Complications Probably Due to Lead Poisoning. Am. Jour. Ophth., vol. 5, no. 6, pp. 468-470, June 1922.

Sellers, Arthur, M.D., D.P.H.: Blood Changes in Lead Workers. Jour. Ind. Hyg., vol. 2, no. 10, pp. 361-367, Feb. 1921.

Sellers, Arthur, M.D., D.P.H.: A Contribution to the Study of Punctate Basophilia in Lead Workers. Jour. Ind. Hyg., vol. 7, no. 4, pp. 145-154, April 1925.

Sharpe, N.C., A.B., M.B.: Report on an Investigation to Determine the Hazard to the Health of Operators Using the Spraying Machine for Painting: The Risk of Lead Poisoning. Jour. Ind. Hyg., vol. 3, no. 12, pp. 378-386, April 1922.

Shie, M.D.: Lead Poisoning: Its Symptomatology and Diagnosis. Jour. Am. Med. Assn., vol. 83, pp. 580-583, Aug. 23, 1924.

Shields, J.B., Mitchell, H.H., and Ruth, W.A.: The Metabolism and Retention of Lead in Growing and Adult Rats. Jour. Ind. Hyg., vol. 21, no. 1, pp. 7-23, Jan. 1939.

Shiels, D.O.: The Ratio of Large to Small Lymphocytes in Persons Exposed to a Lead Hazard. Med. Jour. Australia, no. 25, pp. 847-849, June 20, 1936.

Straube, G., and Beck, H.: Microvanalytic Detection of Lead in Body Liquids. II. Klin. Wchnschr., pp. 356-360, 1939.

Surgeon Gen. Committee, U.S. Pub. Health Service, Washington, D.C.: Report on Tetraethyl Lead, Jour. Ind. Hyg., vol. 8, no. 5, pp. 248-256, May 1926.

Tebbens, B.D.: Atmosphere Lead Contamination from High Temperature Lead Baths. Jour. Ind. Hyg., vol. 19, no. 1, pp. 6-11, Jan. 1937.

Teleky, Ludwig, M.D.: German Literature on the White Lead Question 1921. Jour. Ind. Hyg., vol. 4, no. 3, pp. 100-105, July 1922.

Teleky, L.: A Note on Blood Pressure in Lead Poisoning. Jour. Ind. Hyg., vol. 19, no. 1, pp. 1-5, Jan. 1937.

Vigdortchik, N.A.: Lead Intoxication in the Etiology of Hypertonia. Jour. Ind. Hyg., vol. 17, no. 1, pp. 1-6, Jan. 1935.

Vogt, E.C.: Roentgen Sign of Plumbism: Lead Line in Growing Bone. Am. Jour. Roentgenol., vol. 24, p. 550, Nov. 1930.

Weller, C.V.: Lead Meningo--Encephalopathy. Ann. Clin. Med., vol. 3, p. 604, March 1925.

Wright, Wade: Lead Poisoning. Boston Med. and Surg. Jour., vol. 187, no. 9, pp. 328-331, Aug. 31, 1922.

Wright, Wade, M.D., Sappington, Clarence O., M.D., and Rantoul, Eleanor: Lead Poisoning from Lead Piped Water Supplies. Jour. Ind. Hyg., vol. 10, no. 7, pp. 234-252, Sept. 1928.



